THE TREATMENT OF SOME
ACUTE VISCERAL INFLAMMATIONS

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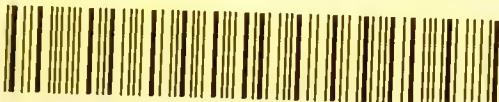
THE TREATMENT OF SOME ACUTE VISCERAL INFLAMMATIONS

AND OTHER PAPERS

"Let me glean, I pray you, and gather after the reapers among the sheaves."

"ἑκάστου τὸ ἔργον ὁποῖόν ἐστιν τὸ πῦρ δοκιμάσει."

THE

TREATMENT OF SOME·ACUTE VISCERAL INFLAMMATIONS

AND OTHER PAPERS

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PREFACE

THE first part of this book consists of three Lectures, delivered before The Harveian Society of London in November 1903. The ideas which they embody are mainly these four :—

 1*st*. The necessity for a more careful, systematic, and repeated determination, by light percussion, of the size of the left ventricle and of the right auricle, in all diseases of the heart and of the lungs.

 2*nd*. The importance of relieving a distended right heart by leeches or moderate venesection.

 3*rd*. The value of the external application of ice as a local remedy in Pneumonia, in Pericarditis, in Pleurisy, in many cases of Appendicitis, and in Acute Nephritis.

 4*th*. The advantage of largely increasing the amount of Sodium Salicylate given in Acute Rheumatism and in Chorea, and the necessity for safeguarding these larger doses by still larger doses of Sodium Bicarbonate.

The papers which follow have been reprinted, partly because they show the gradual evolution of the ideas above enumerated, and partly because they deal with cognate subjects. Among other points, they draw attention to the dilatation of the left ventricle, which is constant in acute and sub-acute rheumatism, and to the still more dangerous dilatation, which is prone to occur in diphtheria and in influenza, also to the acute dilatation of the right auricle, which is met with occasionally in mitral stenosis ; and they suggest appropriate treatment for these several conditions.

They include also a description of the earliest manifestations of rheumatic heart-disease, and of the clinical phenomena of chorea.

Two pathological specimens are also described— a remarkable congenital malformation of the heart, and a rare abnormality of the larynx in an infant.

Finally, it is claimed that a study of the dyspnœa associated with dilatation of the right heart affords proof of the existence of a true reflex from the right ventricle to the respiratory centre, of which there is as yet no evidence from physiological experiment.

My hope is that some at least of these papers may be found to be of permanent value.

D. B. L.

April 1904.

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OTHER PAPERS

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THE TREATMENT OF SOME ACUTE
VISCERAL INFLAMMATIONS

INTRODUCTION

ACUTE inflammation of one or more of the thoracic
and abdominal viscera, usually bacterial in origin,
confronts the practitioner of medicine almost every
day and gives him many anxious moments.   It is
often fatal, cutting short many useful and valued lives,
and thus causing the deepest sorrow and often also
pecuniary disaster to individuals and to families.
When not fatal, it may result in a condition of chronic
disease and incapacity, the effects of which may be
felt as long as the patient lives.

The nature of the diseases thus caused is better
understood than it was twenty years ago, and much
valuable work has been done for their elucidation.
But it cannot be said that a similar advance has been
made in their treatment, or that they are now, to any
considerable extent, less fatal than of old.   Pneumonia
still too often destroys life in a few days, and has even
gained a fiercer virulence by its alliance with influenza,

The acute inflammations of the heart and kidneys still result in shortened, crippled, and distressful lives. There is an urgent need for improvement in the treatment of these acute visceral inflammations. The present methods are largely traditional, and unfortunately these traditions are often incorrect and founded on inaccurate observation.

To advocate measures of different nature is to expose oneself to the risk of being considered a reckless and dangerous innovator ; but this risk must be accepted if any real improvement is to be made. Any such attempt to reform traditional plans of treatment, if it is to be successful, must be founded on long-continued, patient, and careful observation, and on experience of many cases.

In these lectures I desire to state some of the conclusions to which I have been led by my own attempts to improve the treatment of acute visceral inflammations, and to describe the methods which I have employed, more or less fully, during the last sixteen years at St Mary's Hospital, and for thirteen years at the Hospital for Sick Children, Great Ormond Street. The number of beds under my care has been constantly about sixty : thirty for adults, and thirty for children. My first paper on the subject, entitled " Two Cases of Broncho-Pneumonia treated with Bleeding and Ice," was published in 1885. But my interest in the matter is of much earlier date. Twenty-nine years ago, while still a medical student I stood

by the deathbed of one dear to me and dear to many, the mother of ten children, whose health had been excellent until a week before her death, at only forty-nine, from pneumonia. To her memory I dedicate these lectures.

LECTURE I

IT is right to begin with the heart, for the heart is the mainstay of life. More fundamental than the respiration, the digestive, assimilative, and excretory apparatus, or the nervous centres and paths, is the wonderful rhythmic contraction of the cardiac muscular fibre. Cardiac pulsation begins as early as the second day of embryonic life in the chick, very soon after the first appearance of a rudimentary heart, and " long " (say Foster and Balfour) " before the cells of which it is composed show any distinct differentiation into muscular or nervous elements." The pulsation continues throughout the whole of the subsequent life, and physiology now teaches that it is not the cardiac nerves or ganglia, but the automatic rhythmic contraction of the cardiac muscle, which maintains the circulation. When this ceases, nothing can prolong our life. How marvellous—and how little regarded !— is this physiological miracle, the persistent automatic rhythmic discharge of cardiac energy, independent of volition and of consciousness, once in every second or

thereabouts, perhaps for eighty or ninety years! In the course of a long life the heart will have performed three thousand million contractions, and executed an amount of work equal to the raising through a height of one foot no less than $2\frac{1}{2}$ millions of tons. When we have realised these facts we see what a calamity an acute cardiac inflammation or degeneration must be, and how it must almost necessarily shorten life and enfeeble activity.

If the cardiac muscular fibre is to continue its regular rhythmic action, two conditions are essential. First, the wall of the heart must be adequately nourished by the circulation therein of sufficient blood of normal quality, and secondly, the internal tension in the cardiac cavities must be maintained within certain limits, for a normal intracardiac tension is a constant stimulus to the heart, while an excessive tension may dilate it and greatly diminish the force of its contractions. Acute inflammation of the heart interferes with both these conditions. It deranges the circulation of blood in the cardiac wall, and alters the composition of the blood supplied to it. Secondly an acute inflammation of the heart alters the tension in the cardiac cavities, reducing the tension in the left ventricle and increasing it in the right, through the enfeeblement of the ventricles by the inflammatory process, which causes a gradual stagnation of blood in the pulmonary circuit and right heart. But further, an acute inflammation of the heart not only hinders

its efficiency: it tends to injure and destroy the
cardiac muscular fibre itself. The fibres are com-
pressed by the inflammatory products in the inter-
stitial connective tissue, and at the same time poisoned
by toxins in the blood. Hence more or less fatty
degeneration and consequent atrophy of the cardiac
muscle occurs, and a tendency to fibrosis results from
the interstitial inflammation. The fibrous structure
of the heart suffers as well as the muscular. The
valves are swollen and deformed, causing hindrance
to the onward flow of the blood, or regurgitation, or
both. The pericardial covering of the heart may
suffer also, especially when the myocarditis is severe,
for the visceral pericardium is a part of the heart
itself.

The most frequent cause of acute inflammation of
the heart is the rheumatic process, the explanation of
which has now been furnished by the brilliant and
careful investigations of Poynton and Paine. They
have proved that from the tissues of rheumatic
patients after death, as well as from their blood during
life, and from "rheumatic nodules" excised with
aseptic precautions, pure cultures can be obtained of
an organism which, when injected intravenously into
rabbits, can produce in them almost all the various
manifestations of a virulent rheumatism in a child.
This organism is a diplococcus, which grows like a
staphylococcus on solid media, and like a strepto-
coccus in liquid ones. It is a point of much interest

that the cultures always become acid, even though the
culture-medium be alkaline at first.   Poynton and
Paine have demonstrated the existence of these
organisms in the interior of the cardiac valves (with
intact epithelium), also in the interstitial connective
tissue between the cardiac muscular fibres, and in the
pericardial fluid.   The type of inflammation which
they cause in the heart is a fibro-serous one.
Much less frequent, yet more directly fatal, than the
rheumatic diplococcus, as a cause of acute inflamma-
tion of the heart, are the pneumococcus and the
other pyogenetic organisms.   These may cause a
suppurative pericarditis, or a malignant endocar-
ditis; how far they may affect the wall of the
heart is not known.   Suppurative pericarditis is very
much less common than rheumatic : in many of the
cases in which it occurs it is associated with pneu-
monia or with empyema, or is part of a general
pyæmia.   It differs from rheumatic pericarditis in
symptoms and in physical signs, and is often
extremely difficult of diagnosis.   Its treatment differs
greatly from that of rheumatic pericarditis.   The
tubercle bacillus is rarely the cause of pericarditis, and
this is usually of chronic type, and often obscure in its
symptoms and signs.   The diphtheria bacillus causes
by its toxin an intense fatty degeneration of the
cardiac muscle.   The pericarditis of chronic nephritis
is probably toxic in nature, and is usually a terminal
phenomenon.

It is clear, therefore, that the treatment of acute
cardiac inflammation is, in the great majority of cases,
the treatment of an infection with the organism of
acute rheumatism, and we have to consider what
therapeutic agents, general and local, are available to
assist us.

First and foremost, we must determine the value of
sodium salicylate as an internal remedy in acute
rheumatism.   It is believed by some that the value of
salicylate in the treatment of rheumatism is limited
to the reduction of temperature, the relief of pain, and
the diminution of arthritis.   But a very short experi-
ence in the wards of a hospital for children suffices to
show that this view is quite inadequate.   For in the
rheumatism of childhood arthritis is often absent alto-
gether, and pain may be slight, while cardiac disease
is severe.   Yet the good effects of salicylate in
children are quite as well marked as in adults.   The
temperature falls, the general condition improves,
torpidity gives place to a bright expression, and the
cardiac state is ameliorated, the area of dullness tend-
ing to diminish, and the strength of the ventricular
contraction to increase.

Observation of the effects of salicylate in the treat-
ment of acute rheumatism both in children and in
adults has led me to the conviction that it is as truly
antirheumatic as quinine is antimalarial or mercury
antisyphilitic.   But it must, of course, be given in
adequate doses, and its use must not be relinquished

too soon. To an adult, 20 gr. of sodium salicylate
with 40 gr. of sodium bicarbonate should be given
every two hours during the day and every four hours
during the night. To a child of 6 to 10 years old, 10
gr. of salicylate and 20 gr. of bicarbonate may be
given at the same intervals ; this amounts to a daily
dose of 100 gr. of salicylate. After a day or two, 15
gr. of the salicylate with 30 gr. of bicarbonate may be
administered, and if necessary this may be increased
to 20 and 40 gr. respectively ; the total daily amount
of salicylate will then be 150 or 200 gr. Children
require proportionally larger doses than adults, for in
them the rheumatic infection is much more intense.
It is rare for an adult to die from his first rheumatic
attack, but this disaster is by no means uncommon
in childhood. Fortunately children usually bear sali-
cylate well ; in them it seldom causes much vomiting,
often none at all, and they hardly ever complain of
the deafness, tinnitus, and headache which are trouble-
some in adults, nor do they often manifest the mental
symptoms and the tendency to delirium which are
sometimes caused by the drug in later life.

There is, however, one effect occasionally produced
by salicylate which demands special mention, for it
is a symptom of danger, and its occurrence should
lead to an immediate discontinuance of the drug. It
is a marked deepening of the inspirations (apart from
pericarditis), and resembles the " air - hunger " of
diabetic coma. In diabetes an incipient air-hunger

may sometimes be arrested by the administration of large doses of alkalies, and in the only fatal case of salicylic air-hunger which has come under my own observation the salicylate had been taken without any added alkali.   It seems probable that the symptom is due to an action on the respiratory centre caused by an excess of acid.   An addition of sodium bicarbonate in quantity equal to twice that of the salicylate in each dose is usually sufficient to prevent the occurrence of this symptom.

If for any reason, in child or adult, it seems necessary to discontinue the salicylate, it should only be suspended for a few hours ; then it should be given again in a smaller amount, and the dose gradually increased.   It is almost always possible in this way to accustom the patient to the drug, and to get him to tolerate much larger doses than at first produced disturbance.

CASE I.— A woman, aged 25, suffering from mitral stenosis, with high temperature and fresh endocarditis so severe as to suggest a diagnosis of malignant endocarditis, vomited and became delirious on 20-gr. doses of salicylate. By omitting the medicine for a few hours, and then giving smaller doses at first, she tolerated the remedy well, and it was soon possible to increase the amount as far as 30 gr. every two hours. The threatening signs and symptoms disappeared, and when she was discharged only a tranquil stenosis remained.

In severe cases large doses may be necessary, as

much as 30 gr. every two hours for an adult, occasionally 25 or even 30 gr. for a child. It is very desirable to continue the use of the drug for a considerable time after the subsidence of the rheumatic symptoms. Too early discontinuance of the remedy, or a too great reduction of the dose, is often in adults, and still more frequently in children, quickly followed by a relapse. Too great timidity in the employment of salicylate may cause an adult patient additional pain which might have been avoided; in a child it may be the cause of a cardiac disaster.

CASE II.—A boy, aged 5, with a greatly dilated rheumatic heart, was treated with 8 gr. of sodium salicylate and 16 gr. of bicarbonate every two hours for sixteen days, and manifested decided improvement. The frequency of the dose was then reduced to every four hours, that is, the daily dose of salicylate was reduced from 96 gr. to 48 gr. Five days later, while the smaller dose was still being taken, a relapse occurred, with increased cardiac dilatation, and this led to his death three weeks later.

Case III.—A woman, aged 52, suffering from rheumatic arthritis in many joints, and with loud pericardial friction, was treated on 10th July 1903, with 200 gr. of sodium salicylate daily, with 400 gr. of sodium bicarbonate, and an icebag was applied over the heart. Four days later the arthritis had disappeared, and the rub was much less loud, though the temperature had fallen only from 103.5° to 102°. But she complained so much of headache, deafness, and tinnitus that the medicine was discontinued altogether for two days. On 16th July it was again given, but in

smaller amount—120 gr. of the salicylate and 240 gr.
of bicarbonate.   The temperature continued to fall
for two days longer, and on the 18th was only 99°;
but on the 19th, five days after the temporary sus-
pension of the remedy, it rose again to 100.2°, and
continued at this height on the 20th, with a further
rise on the 21st to 101.8°.   It seemed evident that a
relapse of rheumatism was coming on, which the
amount of salicylate and bicarbonate then being
taken was insufficient to prevent.   With a view of
testing the question whether it was the salicylate that
was really necessary, or whether an increased dose of
the alkali would suffice, the daily dose of this was
raised to 360 gr., the amount of salicylate being still
only 120.   The temperature rose on that day to
103.4°, and the next day (23rd) to 103.9°, and fresh
rheumatic pain was then felt in the right hand.   On
the 24th the metacarpo-phalangeal joints of this hand
were red and swollen.   It was clear that the increase
in the amount of alkali had failed to meet the need.
The dose of salicylate at first given was therefore
resumed, 200 gr. daily with 400 of bicarbonate.   Next
day (25th) the redness and swelling of the fingers had
subsided, but on the 26th a fresh pericardial rub was
heard, which was louder and harsher on the 27th.
On the 28th it was very much less, and the tempera-
ture had fallen to the normal.   From 26th July to
8th August she took 160 gr. of salicylate and 320 of
bicarbonate; but as the temperature again rose to
99.5° on the 8th the doses were again increased to
200 gr. and 400 gr.   There was no further recurrence
of rheumatism, and she now took without difficulty
the same doses as had at first caused disturbance.
The temporary omission of the medicine and the
smaller subsequent dose were clearly responsible for
this fresh rheumatic outburst in joints and peri-
cardium.

The employment of salicylate in sufficient doses for a sufficiently long period is much hindered by a tradition current among us that this drug is a "cardiac depressant." This doctrine has been maintained by some physicians of great authority as clinical observers, and especially they have taught that when pericarditis occurs in a patient suffering from rheumatic fever the salicylate ought to be stopped lest it should depress the heart still more. Now it is clear that a belief of this kind must have been founded on experience of a tendency to failure of the pulse and weakness of the first sound of the heart observed in rheumatic patients while taking salicylate. But one may fairly ask whether it is quite certain that such occurrences are really the effect of the drug, whether they may not be due to some other cause? In the first place, they may conceivably be due to impurities of the drug as administered. Especially in the early years of its use, when a sudden demand arose for large quantities of the new remedy, it is quite likely that adulterations due to imperfect methods of preparation may have caused toxic symptoms, the memory of which remained in the minds of those who witnessed them, and gave to the drug an evil reputation which has clung to it ever since. The sodium salicylate now obtainable is carefully prepared and purified, and is free from impurities of cresols, etc. That obtained from oil of winter-green is more than ten times as costly,

so that its employment in large doses is impracticable.

But there is another and a more important fact which has been the main cause of the belief in the depressing effect of salicylate. It is unquestionable that rapid extension of the dullness of the left ventricle with enfeeblement of the first sound and of the pulse may occur in rheumatic patients who are taking salicylate. But is this due to the drug or in spite of it? Has it caused these symptoms or merely failed to prevent them? Can they be due to the disease itself?

Acute rheumatism produces, apparently in all cases of the disease, more or less dilatation of the left ventricle, with a tendency to diffusion and weakening of the impulse and enfeeblement of the first sound. I drew attention to this fact in a paper on acute dilatation of the heart in rheumatic fever, followed by a joint paper with Dr Poynton on the same phenomenon as observed in the rheumatism and chorea of childhood, both published in the *Transactions of the Royal Medical and Chirurgical Society* for 1898. These observations have not, so far as I am aware, been controverted, and they have been confirmed by subsequent experience.

In the mildest case of subacute rheumatism the dullness of the left ventricle hardly ever fails to reach the nipple-line; usually it extends a fingerbreadth beyond it. It may attain to two fingerbreadths to the left of

the nipple-line, without any murmur, and it may
return to the normal as the attack subsides.   This
dilatation is due, no doubt, to the toxic action on the
cardiac muscle of the toxin produced by the rheumatic
organisms, frequently also to the actual presence in
the heart-wall of the organisms themselves.   We can
better appreciate the probable effect of the toxin by
remembering Dr Gaskell's experiments, recorded in
the third volume of the *Journal of Physiology*.   Dr
Gaskell found that a dilute solution of lactic acid
caused relaxation and "extreme dilatation" of the
frog's ventricle, and finally diastolic standstill; it
caused relaxation also of the vessels.   On the other
hand, a dilute solution of sodium hydrate caused
gradual progressive contraction of the ventricle until
it remained persistently fully contracted, and a similar
contraction of the arterioles.   He investigated also
the action of various drugs, and found that some of
them acted upon the ventricle like soda, others like
lactic acid.

It seems almost certain that the toxin produced by
the rheumatic cocci acts upon the ventricle in a
manner similar to that of lactic acid ; and it is of
interest to remember that cultures of these organisms,
even in an alkaline medium, always become acid ;
also to remember the belief of many former clinical
observers that the poison of acute rheumatism is
lactic acid itself.   Important evidence on this question
has recently been brought forward by Dr Ainley

Walker and Mr Ryffel in a report to the Scientific
Grants Committee of the British Medical Associa-
tion.*   They state that the " micrococcus rheumaticus "
produces *formic* acid in very considerable quantity,
and that this acid is not only present in the filtered
cultures of the organism, but can also be extracted
from the bodies of the micro-organisms themselves.
From these, after repeated washings to remove
adherent traces of the culture - fluids, they have
obtained appreciable amounts of formic acid.   It
follows that the processes concerned in its production
proceed within the bodies of the micro-organisms
themselves.   From a litre of culture-fluid they were
able to obtain about half a gram of formic acid.   In
addition to formic acid, the cultures and also the
washed micrococci contain at least one other acid of
the fatty acid series, probably acetic.

They also succeeded in obtaining formic acid from
the tissues of a rabbit suffering from acute arthritis
due to the inoculation of this micro-organism.

They state, also, that while from normal urine
formic acid is absent or occurs in traces only, formic
and probably another fatty acid are present in appre-
ciable amounts in the urine during the course of acute
rheumatism, and they have some evidence that under
the salicylic treatment of rheumatism the amount of
formic acid in the urine is reduced.

With regard to the origin of the formic acid, they

* *British Medical Journal*, 19th September 1903, p. 659.

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believe it to be formed by the micro-organism by the oxidation of sarcolactic acid into acetic and formic acids according to the equation :

$$CH_3 \, CHOH \, COOH + O = CH_3COOH + H.COOH.$$
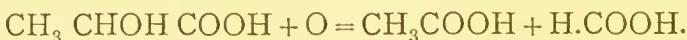
They add two facts of much importance. They have observed that the micrococcus rheumaticus has a hæmolytic action upon red blood-corpuscles greater and more rapid than that of any other streptococcus which they have yet examined, which explains the rapid and considerable anæmia of acute rheumatism. They have also succeeded in isolating an albumose from cultures of the organism grown in albuminous fluids free from albumoses, which on injection into animals (guinea-pigs and rabbits) rapidly produces marked pyrexia, and leads to an increase of temperature of three or more degrees Fahrenheit, reaching on one occasion 105°.

These observations of Dr Ainley Walker and Mr Ryffel are a justification of the ancient practice of treating acute rheumatism with alkalies. Before the specific action of the salicylate was discovered the alkaline treatment of rheumatism was the most successful, though at that time potassium salts were chiefly used and in comparatively small quantities. But sodium salts can be given much more freely, and Dr Gaskell's observations on the frog's heart seem to indicate that they also tend to cause contraction of the ventricle. I think that there is some clinical

evidence that this is really the case when they are freely employed in rheumatism.

If, then, the rheumatic toxin always tends, more or less, to dilate and enfeeble the left ventricle, it is not surprising to find that in some cases a "cardiac depression" may manifest itself somewhat suddenly, after the disease has lasted for some days, or even a week or two. If the patient is taking salicylate when this occurs, the drug is almost certain to receive the discredit of symptoms really due to the disease itself. But strong evidence as to their actual cause can be adduced from analogy, for a similar acute dilatation and enfeeblement, but of a much more dangerous kind, is met with in some other diseases, notably influenza and diphtheria.

I pointed out, in an address on acute dilatation of the heart in diphtheria, influenza, and rheumatism, delivered before the Manchester Medical Society in 1900, and published in the *British Medical Journal* for 5th January 1901, that in all these diseases an acute cardiac dilatation is not uncommon, and that it may cause fatal syncope in diphtheria and in influenza.

The symptoms before death are pallor, sometimes vomiting, and feeble (perhaps irregular) pulse. The physical signs are a rapid increase in the heart's dullness to the left, which may amount to an additional fingerbreadth in a few hours, diffusion and weakening of the impulse, with weakening of the first sound and

of the pulse-wave. After death extensive fatty degeneration of the cardiac muscle is found. The symptoms of an acute dilatation in rheumatism are less serious than those of a similar condition in diphtheria and in influenza, probably because there is less destruction of the cardiac muscular fibre. The acute dilatation of rheumatism rarely, if ever, produces a fatal syncope, but it may give rise to marked pallor and to obvious changes in the pulse. In diphtheria an acute dilatation may occur at any time during the six weeks following the onset of the disease. In rheumatism it may occur as late as two or three weeks after the onset, perhaps later.

Case IV.—A woman, aged 29, was suffering from her first attack of rheumatism. It was of a subacute type, with but little arthritis; the cardiac dullness extended to the nipple-line but not beyond it, and there was no murmur. She was treated with sodium salicylate. After she had taken this for a fortnight with benefit, the house-physician one day observed that the pulse was unusually weak. He percussed the heart at once, and found that the dullness of the left ventricle now extended one fingerbreadth beyond the nipple-line, and a short systolic murmur could be heard. I confirmed these facts on the following day. Here was a case in which it might have been suggested that the salicylate had caused cardiac failure. But instead of omitting the drug I ordered it to be given more frequently, and placed an icebag over the heart. Immediate improvement followed, and in a few days the cardiac dullness was normal, and the faint murmur had disappeared.

In diphtheria it cannot be doubted that an acute cardiac dilatation is due to the disease itself, and not to any drug that has been administered.   I am convinced that in rheumatism also it is the disease itself which is responsible for cardiac failures, and that they are not due to the salicylate.   Indeed, they are probably in many cases an indication that an insufficient amount of the drug has been given.

If more care were taken to determine the exact size of the left ventricle by careful light percussion at the commencement of treatment and daily afterwards, an acute dilatation would be at once detected.   There is little difficulty in this determination if the fingers only and not any " pleximeter " are used for percussion, if the part of the finger percussed is the terminal phalanx, and the other phalanges are not allowed to rest on the thoracic wall, and if the percussion-stroke is always light.   It is a primary duty to determine this matter with care whenever a practitioner is called on to attend a case of diphtheria, of influenza, or of rheumatism.   It is, I believe, the neglect of this which has caused salicylate to be considered a cardiac depressant, and thus has done much mischief to rheumatic patients, for it has deprived them of a necessary drug.

If, then, salicylate is not a cardiac depressant, and if it is genuinely antirheumatic, it is surely specially necessary in order to counteract the most deleterious action of rheumatism—its tendency to cause cardiac

inflammation. The worst type of rheumatic carditis is associated with pericarditis, and it is here that the drug is most urgently needed. To withhold salicylate from a rheumatic patient, child or adult, unless some equally efficacious medicine can be substituted for it, is, in my judgment, to do the patient a grievous injury. In medicine, as in ordinary life, sins of omission are more lightly regarded than sins of commission, but they may be quite as disastrous.

In support of my plea for the employment of larger doses of salicylate in the treatment of rheumatic conditions, I may mention that I have lately found great advantage in the administration of large doses of salicylate and bicarbonate in many cases of chorea. The small doses of these remedies usually given in this disease (50 to 60 gr. a day) are far too small. If the amount of salicylate given at first, in divided doses, be 100 gr. per diem, and this be rapidly increased to 150 gr., and if necessary to 200, very remarkable improvement often occurs, as I showed at the discussion on chorea at the Swansea meeting of the British Medical Association. After this discussion, a fact of interest was mentioned to me by Dr McVittie, of Dublin, and he kindly allows me to relate it now. Through the mistake of a chemist, 2-drachm doses of sodium salicylate were taken by a patient three times daily for a week, with much benefit, and without the occurrence of any unpleasant symptoms. Encouraged by this observation, Dr McVittie pre-

scribed for another patient suffering from chronic
rheumatism, powders containing 2-drachm doses of
salicylate, to be taken three times a day.   The chemist
to whom the prescription was taken thought that a
mistake had been made, and distributed 2 drachms
of the salicylate, as a total amount, among the twelve
powders.   The patient took these powders, and was
no better.   Then he had the prescription made up by
another chemist, who put into the powders the full
dose ordered.   In a. week the man was well.   In two
subsequent cases of chronic muscular rheumatism Dr
McVittie observed complete relief of all pain follow
the use of these powders, and no unpleasant symptoms
were produced.

My conclusion is that sodium salicylate has
certainly a marked antirheumatic action, that a con-
siderable daily dose of the drug is essential to success,
that each dose should be accompanied by twice as
large a dose of sodium bicarbonate, and that the
joint action of the two drugs should be maintained
for some time after the subsidence of the rheumatic
symptoms.

We have, therefore, in the free administration of
sodium salicylate and bicarbonate an adequate
method of meeting the causal indication in the
treatment of rheumatic inflammation of the heart
and pericardium.   It remains to inquire whether we
have also any means of directly diminishing the
inflammatory action by local applications.   The use

of blisters over the precordial region was at one time
a favourite method of treatment, and it is at least
conceivable that the counter-irritation of the overlying
skin may tend to diminish the fullness of the vessels
in an inflamed pericardium.   But it is not desirable if
it can be avoided to add to the sufferings of a patient
who is seriously ill, and the local effect of a blister
greatly impedes the necessary examination of the
heart.   Leeches applied to the cardiac area have a
distinctly beneficial effect.   They probably have some
direct influence in draining the pericardial vessels,
and they certainly cause a diminution of pressure in
the right auricle, which is often of the greatest service.
But this latter effect can be equally well obtained by
applying them to any other part of the thoracic wall,
thus lessening the supply of blood through the azygos
vein to the right auricle, and the subsequent examina-
tion of the heart will then not be impeded.   The
most convenient place for their application is over
the lower anterior part of the right chest below the
nipple-level ; it is always possible to arrest the flow of
blood by pressure against the firm resistance of the
ribs.   But if the leeches are applied over the soft
parts at the epigastrium there may be very consider-
able difficulty in arresting the flow.

The persistent application of cold to the precordial
region by means of an icebag is a powerful means of
repressing cardiac inflammation.   In rheumatic peri-
carditis its effect is unmistakable, and often very

striking. I have employed it in nearly every case
under my care during the last eleven years, and
have found it extremely useful. At first I was very
cautious in using it, only applying it for a short time,
lest the cold should further depress a heart already
hampered by the pericardial inflammation. What
led me to employ it was the observation that where
an icebag had been applied for some hours to a
pneumonic lung, it was usually found that the dull-
ness at that spot was less intense, and that there was
a better air-entry, indicating that the vascular con-
gestion of the lung beneath the icebag had been
lessened. I found also that the symptoms and
physical signs of subacute inflammation of the appen-
dix vermiformis were rapidly relieved by an icebag,
and that the same was true of sciatic neuritis of
recent origin.

In all these three cases the direct local influence of
the icebag in repressing subjacent visceral inflamma-
tion seemed undeniable, and it occurred to me that
possibly it might have the same beneficial influence
in pericarditis. I soon found that the inference was
correct, and that there was no danger of collapse if
two precautions were kept in mind.

One of these precautions is that the patient be
made quite warm, if necessary by hot-water bottles,
before the icebag is applied to the heart, and that the
warmth be maintained during the whole period of its
application. The second precaution is that the right

auricle shall not be allowed to be overdistended. A few leeches, applied as already recommended, are sufficient to fulfil this condition, and it is generally advisable to apply them before the ice is used.

If these two conditions are secured, the icebag may be used without fear in a case, however severe, of rheumatic pericarditis. With these precautions the local use of ice, far from depressing the heart, has actually a tonic action. The feeble and diffused impulse of the left ventricle becomes localised and stronger ; this effect is often very noticeable after the icebag has been used continuously for some days. The friction-sound soon becomes less harsh and is heard over a smaller area of the precordium. This is not due to increased exudation, for the area of precordial dullness tends to diminish. If at the same time large doses of sodium salicylate and bicarbonate are being administered, the improvement may be very rapid, and the diminution of the dullness remarkable. It must be remembered that in rheumatic pericarditis the effusion of fluid into the pericardial sac is only a minor factor in the increase of the dullness ; this is mainly caused by dilatation of the heart. It is necessary to insist on this fact, for it is not yet generally recognised. Rheumatic dilatation of the heart without any pericarditis may give rise to an outline of precordial dullness, which is practically a triangle with curved sides and with its apex upwards. When pericarditis is present also, a moderate amount

of pericardial effusion tends to accumulate behind the dilated heart in the recumbent position, and does not much influence the shape of the dullness. The greatest effect of a moderate pericardial effusion on the dullness is produced in the second left intercostal space, in which a definite lateral increase of the dullness suggests strongly the existence of fluid in the pericardial sac. A large pericardial effusion will no doubt extend the cardiac dullness further in all directions, but it remains true that in rheumatic pericarditis the chief factor in the enlargement of the dullness is dilatation of the heart.

The outline of the precordial dullness on the right side is mainly indicative of the state of distension of the right auricle. This statement also needs to be specially insisted on, both on account of its value as a suggestion for treatment and because it is as yet by no means universally acknowledged that the condition of the right auricle can be determined by careful percussion, its enlargement in disease of the lungs or left heart watched and recorded, and its diminution after leeches or venesection easily appreciated. In the normal heart the dullness of the right auricle always extends about one fingerbreadth (rather less in a child) to the right of the sternum in the fourth intercostal space. This can easily be verified by any one who avoids all artificial pleximeters and percusses with a light hammerstroke, with the flexed terminal phalanx of a finger of his right hand, on the terminal

phalanx of a finger of his left hand, firmly applied, taking special care to keep the other phalanges of the percussed finger and the whole of the rest of the left hand away from contact with the thoracic wall. Unless this precaution be adopted, pulmonary or gastric resonance is brought out at the same time as the truly cardiac, and confuses the result. Lightness of percussion-stroke also assists in obtaining the true cardiac note, for though the right auricle is overlapped by lung, the auricle with its contained blood gives a dull note which the thin overlying lung does not alter much, and at the border of the auricle there is a definite alteration in the note which careful percussion easily elicits.

In the normal heart it is usually difficult to detect any dullness in the third right space, but it is always definite and considerable in the fourth. In the fifth space the partial liver-dullness complicates the result. It is of the greatest practical importance that the extent of the dullness in the fourth right space should be carefully determined in every examination of the heart, and no such examination is anything but fallacious unless this point has been carefully investigated.

In chronic disease of the lungs or left heart, as in emphysema, and in mitral stenosis, the right auricle is always dilated, and its dullness may extend to two fingerbreadths in the fourth right space : it is then easily detected in the third space also, and may there amount to a half or one fingerbreadth. In extreme

cases it may reach even three fingerbreadths in the
fourth space, one and a half in the third and half a
fingerbreadth in the second.

In acute pulmonary disease, as in pneumonia, and
in severe bronchitis, an enlargement of the right
auricle may be detected, often on the third day of the
illness, sometimes even on the second. Acute rheu-
matic carditis affects at first and chiefly the ventricular
muscle, and causes distinct enlargement of the cardiac
dullness to the left, but in a first attack it is difficult
to detect any affection of the auricle. But where
pericarditis is present there may be marked increase
of the auricular dullness. And this observation is of
great importance from the point of view of treatment.
For whenever the right auricle is acutely distended
there is hurried breathing. This is usually the
explanation of the dyspnœa which is acknowledged to
be one of the symptoms of pericarditis; and this
dyspnœa is a sign that the right heart is overstrained.
In pericarditis, then, if there are rapid and deep
inspirations and the dullness of the right auricle in
the fourth space is found to amount to two finger-
breadths, the need for some removal of blood is
evident. Sometimes leeches applied over the lower
ribs on the right side will suffice, but in a severe attack
in a robust subject venesection may be highly advisable.
I have seen it give very great relief. The same is
true in the acute distension of the right auricle in
pneumonia, in acute exacerbations of chronic

bronchitis, and often in the later stages of mitral stenosis.   Dyspnœa and the physical evidence of dilated right auricle are the indications for bleeding, which in these circumstances is a remedy of priceless value, capable of giving immediate relief impossible by any other means.

The first step, then, in the local treatment of rheumatic pericarditis is the accurate determination of the amount of dullness in the fourth right interspace, and the relief of a distended right auricle by leeches, or in an adult by a venesection removing from 4 to 8 oz. of blood.   It may be well to follow this by cardiac stimulation by means of a hypodermic injection of strychnine.   Two minims of the official solution may be used for an adult, $\frac{1}{2}$ or 1 minim for a child.   The injection may be repeated after three hours.   Meanwhile, at least two hot-water bottles should be placed in the bed so as to make the patient's lower limbs thoroughly warm.   When this has been accomplished (and not before), an icebag large enough to cover the whole precordial region and about half filled with small lumps and fragments of ice should be gently laid over the heart as the patient lies on his back.   If there is much tenderness the icebag may be at first suspended so as only just to touch the skin, but the anodyne effect of the cold will soon allow of its more thorough application.   The icebag will not by its weight increase the difficulty of breathing if the right auricle has been first relieved.

The patient soon recognises the beneficial in-
fluence of the ice. Even young children, after the
first few minutes, easily become accustomed to the
cold, and will not consent to its removal. When it
has been removed for an interval they will some-
times ask that it may be reapplied. A girl of
11 under my care at the Hospital for Sick
Children said, when the nurse removed the icebag
for a fresh supply of ice, " May I have my icebag
back again ? " When asked why she wished to
have it, she replied, " Because it eases the pain."
A boy of 7 at St Mary's refused to allow his icebag
to be taken away. To the inquiry, " Why not ? "
he answered, " Because I like it." To the further
inquiry, " Why do you like it ? " he replied, em-
phatically, " Because I *do*."

The chief difficulty about the application of the
icebag is to secure it from shifting its position when
the patient turns in bed. This may be accomplished
by passing the screw-top of the icebag through a hole
in a binder which is passed loosely round the chest
and secured by safety-pins. Sometimes it may be
desirable to prevent its slipping downwards by secur-
ing it by a light bandage from behind the patient's
neck. An excellent method used at the Hospital for
Sick Children is by a vest made of domett, with arm-
holes and a third hole over the cardiac region, and
fastened round the neck with a tape, and below the
icebag by a safety-pin.

The outline of the precordial dullness should be marked on the skin in blue for the guidance of the nurse. The icebag will require to be refilled about every hour and a half, the hot-water bottles every three hours.

It is well to have a second icebag in use, so that it can be filled and applied as soon as the first is removed. In filling the icebag, be careful to press out the air as much as possible before screwing on the top. The hot-water bottles should not all be removed from the bed at the same time, unless others are at once substituted. The patient's temperature should be taken every two hours, also his pulse-rate and respiration-rate, and the result recorded on a chart. The nurse must see that the icebag does not leak, that its top is firmly screwed down to the " washer," and that it is surrounded by cotton-wool or a soft towel to absorb the moisture from the air which tends to condense on its surface. Nothing should intervene between the icebag and the patient's skin.

When the nursing can be thoroughly trusted, as in a hospital, the icebag may often, with great benefit to the patient, be applied continuously for many days, provided that the condition of the right auricle be carefully watched, and the lower limbs be kept thoroughly warm. Occasionally it may be wise to remove it for a few hours during the night, especially between midnight and six o'clock in the morning. If the nurse has had little experience of children, or is

not accustomed to the use of the icebag as here recommended, it will be safer to apply it only during the daytime. If no skilled nursing is available, the treatment may still be used during the daytime if the mother is intelligent and careful, and the practitioner explains to her the use of the clinical thermometer, and tells her precisely what to do. If the practitioner is in doubt whether the icebag ought to be continued, let him order its removal for one hour, and thereafter its application for two hours, and so on.

It is sometimes possible to continue the application of ice to the precordial region even though the temperature have fallen below the normal. A child under my care at St Mary's some years ago, whose pericarditis had improved under this treatment was found one evening to have signs of pneumonia at the base of one lung. Twice before I had met with this in young children suffering from pericarditis and treated with ice ; in each case when the pneumonia appeared I removed the icebag from the heart, fearing that it might be doing harm. Both patients died. When in this third case I again found pneumonia appearing in a case of pericarditis treated with ice, I determined to persevere in my plan of treatment and to push it further. Two more leeches were applied, and a second icebag was placed over the inflamed lung, that over the heart being retained. I should hardly have ventured on this but for the kind willingness of my house-physician, Dr Gordon (now

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medical superintendent of the City Fever Hospital, Manchester), who remained by the child's bedside and was able to keep the two icebags in position during the whole night, though the temperature was at times subnormal. When I saw the child next morning the signs of commencing pneumonia had disappeared and recovery was uninterrupted. This case proved to me that with adequate care it is possible to use the ice in pericarditis when the temperature is low. In the later stages of a pneumonia, however, this is not the case.

The treatment of rheumatic pericarditis by large and frequent doses of sodium salicylate and sodium bicarbonate, with the local application of ice over the heart, is amply justified by its results. Cases treated without ice, and with inefficient medication, are very apt to linger in recovery, and to result in a permanently dilated and crippled heart. Relapse of rheumatism frequently occurs, and ends the case in a year or two. Even apart from a probable relapse, the heart is often so severely injured that symptoms of failure of the circulation develop early. Treatment of the kind here advocated greatly diminishes the tendency to rheumatic relapse; it checks the inflammation, increases the vigour of the muscular fibre, and diminishes the dilatation, thus enormously assisting the forces that make for repair. The practical difference to the patient is often a difference of many years of life.

In the milder cases of rheumatic carditis, those in which there is no evidence of pericarditis, similar treatment (without the bleeding) may be used with great advantage, though the effect is less striking. Since in every case of rheumatism, acute or subacute, there is dilatation of the left ventricle, as may easily be proved by careful percussion, it is clear that there is always some affection of the cardiac wall, either toxic or inflammatory. The cardiac muscle is weakened, as is proved by the altered first sound, the diffused impulse, and the enfeebled pulse-wave. A murmur indicative of endocarditis may or may not be present. Here, then, as really though not so forcibly as in pericarditis, there is a call for antirheumatic medication and for means of repressing local inflammation. I believe that if this method of treatment were universally adopted in the slighter attacks of rheumatism it would very greatly diminish the number of cases of mitral regurgitation and stenosis in adults, and would prevent an enormous amount of cardiac misery.

Suppurative Pericarditis.

When we pass from acute cardiac inflammation caused by the rheumatic diplococcus to the inflammation caused by the pyogenetic cocci, we find conditions differing greatly from the rheumatic, in physical signs, in concomitants, in prognosis, and in treatment. When these cocci, notably the pneumococcus and

streptococci, infect mainly the endocardium, various types of malignant endocarditis develop. With these I have no concern in these lectures, though I may mention that in a child under my care, a fatal heart-disease which absolutely resisted salicylates was found to have been caused by an exuberant endocarditis around the tricuspid orifice, the mitral valve being quite healthy, and that from this endocarditic growth only the pneumococcus could be obtained by culture.

Suppurative pericarditis is very much less common than rheumatic, but it resembles the latter in mainly affecting children and young adults. It is generally associated with suppurative inflammation elsewhere, especially with empyema. The pus in the peri-cardium is often in small amount, and often loculated by adhesions; the diagnosis is then extremely diffi-cult. For there is usually no friction-rub audible, and in suppurative pericarditis the heart is little, if at all, dilated. The most trustworthy indications appear to be an extension of dullness in the second left space, and a rapid, feeble pulse, the heart-sounds becoming feeble and distant. Where the amount of pus in the pericardium is considerable, as in a case of Dr Cheadle's, which he kindly allowed me to examine (a child with pus in the right pleural cavity, who did not improve much after the empyema had been drained), it may be possible to ascertain definitely a considerable extension of the precordial dullness. In Dr Cheadle's case, Mr Pepper made an incision into the dilated

pericardial sac and evacuated a considerable quantity of pus. The child lived for a week or two afterwards.

Suppurative pericarditis, with a moderate amount of pus, is usually mistaken for loculated empyema of the left side. It is too common for the truth to be discovered only in the post-mortem room.

In three cases in which I suspected a small amount of pus in the pericardium, my colleague, Mr Kellock, made an exploratory incision into the pericardial sac. In the first and second of these cases the diagnosis proved to be mistaken. In the third it was correct, but the operation was useless, for adhesions to the ventricular wall forbade further exploration. In all three cases no harm was done by the incision, which healed by first intention. In the autopsy on the third case it was found that there were loculi of pus around the base of the heart, but that over the anterior surface of the ventricle there was adhesion. A fourth case, in which it seemed to me that pus in the pericardium was very probable, and in which I was considering the question of operation, gradually and progressively improved, and finally recovered.

LECTURE II

PNEUMONIA

THE disease which we call "pneumonia" consists in an invasion of the lungs and air-passages by the micrococcus lanceolatus or pneumococcus, its rapid multiplication in this warm culture-chamber, together with the efforts, more or less successful, which the human organism puts forth to resist the advance of the invading foe and to destroy it. The invader often lies in wait in the mouth of its unsuspecting host, for it can be isolated from the saliva of many healthy persons, as was first proved by Sternberg. Life to such persons is, as it were, a feast of Damocles, with a sword hanging by a hair over their heads. Too often the sword falls and the feast of life is over. A chill depresses the vital power of resistance, and the microbe runs riot in the pulmonary alveoli. Or the way is prepared by other infections, notably by the influenza bacillus, and as the influenzal infection subsides the pneumococcal infection develops, and is then of unusual virulence.

The conflict between the invaders and the tissues may begin at one definite spot, as is usual in adults, or it may begin at the same time in several places, as is more frequent in early life. It tends to spread rapidly over a large area of pulmonary territory, and soon becomes a pitched battle, in which all the powers of the defenders are sorely taxed to provide an adequate resistance. The blood-supplies necessary for the very life of the defenders are interrupted by the stagnation in the lungs. The fight grows so intense that sleep becomes impossible, for all the vital energies are engaged. The invaders do not scruple to use poisoned weapons, and their poisons, and even some of the enemy themselves, make their way into the blood and poison the brain.

When the invaders are comparatively few or feeble, and when the resistance is powerful, the battle, after lasting from three to seven days or even longer, ends in favour of the defence, often with dramatic suddenness. The precise explanation of this remarkable phenomenon is still unknown, but the fact is familiar. For some reason or other the attack has failed, and all that remains for the exhausted defenders is to dispose of their dead foes, to clear away the débris of the battle, to cleanse the battle-ground, and to restore the *status quo*. We have been taught to expect this happy issue of the struggle in children of more than 2 years of age, and in young adults with healthy hearts, normal livers, and sound kidneys, and it is

certainly in such patients that the outlook is most
hopeful.   But the advent of the influenza bacillus has
considerably reduced the confidence which formerly
we felt with regard to the issue of a pneumonic attack
in the earlier half of life, and most of us have sorrow-
ful reminiscences of friends, strong and healthy, cut
off by influenza-pneumonia in the very prime of early
manhood.

Often, alas! the invaders are too powerful for the
resistance, and the patient succumbs.   In the first and
second year of life, and again after 35, the death-rate
from pneumonia is very considerable.   Dr Hector
Mackenzie says * that pneumonia " *causes very nearly
as many deaths as enteric fever, diphtheria, small-pox,
measles, and scarlet fever put together*," and he quotes
from the reports of the Registrar-General the follow-
ing figures showing the annual rate of mortality from
pneumonia per million living at different ages :—

All ages		1066
From birth to 5 years .	.	3668
,, 5 to 10 years .	.	299
,, 10 to 15 ,, .	.	119
,, 15 to 20 ,, .	.	201
,, 20 to 25 ,, .	.	301
,, 25 to 35 ,, .	.	494
,, 35 to 45 ,, .	.	833
,, 45 to 55 ,, .	.	1157
,, 55 to 65 ,, .	.	1762
,, 65 to 75 ,, .	.	2596
Over 75 ,, .	.	3187

* *Practitioner*, 1900, p. 36.

He calculates that in a population of 30 millions more than 220,000 persons in each year are affected with pneumonia, and nearly 32,000 annually die from this disease. When the vitality is feeble, when the heart, liver, or kidneys are damaged by previous disease, when there are old pleural adhesions, when the lungs are fibroid or emphysematous, when the patient is chronically poisoned with alcohol or tobacco, and when septic influences are at work, there is but a poor chance of recovery. In many morbid states pneumonia ends the scene. Thus it becomes almost impossible to construct statistics which could give us accurate information as to the ordinary mortality of pneumonia, or as to the results, good or bad, of any proposed method of treatment. But the outstanding fact is this—that *every pneumonia is a fight for life.* If we realise this, we shall never neglect its earliest stages ; we shall not fold our arms in careless or un-observant "expectation," letting slip our opportu-nities of helping the patient in his struggle until we are confronted with a disaster which we have done nothing to avert. Can we not do something to pre-vent the loss—how frequent to-day !—of the most important and valued lives, of fathers and mothers of families, of men and women in responsible and influential positions, whose continued life and activity may be of tremendous importance to their relatives and friends, to the community in which they live, perhaps even to the whole nation ?

If we are to succeed in this, it must be by the most unremitting attention, the most minute and careful observation, and by the exercise of a sound judgment founded upon previous experience. There is no disease which makes more demands upon the practitioner than pneumonia, there is none in which his action or his inaction, his wise interference or his inertia, may more affect the issue. Some cases will no doubt defy his best efforts, some would struggle through without his aid, but in a large number he can afford very great relief, and in many he may have the intense satisfaction of having certainly saved a life. But to accomplish this he must be willing to spare neither time nor trouble. He must visit his patient at least twice daily, at the most critical times he must, if possible, see him thrice in the twenty-four hours. The struggle is short and sharp. Every fresh move of the enemy must be at once detected and counteracted. The entire position of the attack and the defence must be estimated at every visit, and coming dangers must be anticipated and provided for.

In the treatment of pneumonia one thing is, unfortunately, still lacking to us. We have no specific medication. No drug is yet known to us which has an action on the pneumococcus such as that which the salicylates manifest in rheumatic infections, or that which quinine exhibits in overcoming the malarial hæmatozoa. Yet our reminiscence of the days when

rheumatism had to be treated without salicylates should encourage us in the hope that some day we may possess a specific remedy. Neither from serum-therapy is there any present prospect of assistance. Yet here also it would be rash to predict what the future may have in store for us.

But we have to consider the position as it exists to-day. If we cannot overwhelm the foe by ordinary medication or by the subtle influence of an antitoxin, we may at least help the patient to fight his battle, and in more ways than one intervene actively for his relief. The problem is essentially how to keep him alive until the battle is over. A prolongation of life for forty-eight hours will often turn a threatening defeat into a glorious victory.

1. *The Right Heart.*

How, then, does pneumonia tend to kill? Usually by cardiac failure. But this is a failure, not of the left side of the heart, but of the right ; not by syncope, but by asphyxia ; not through enfeeblement of the left ventricle, but through paralysing overdistension of the right.

It is remarkable how little dilatation of the left ventricle can be detected in pneumonia. Of course the pulmonary stagnation tends to diminish the tension in the left ventricle ; but if the pneumococcal toxin were as injurious to the ventricle as the rheu-matic, an increase of the cardiac dullness to the left

would be unmistakable. In influenza such a toxic
dilatation may be often observed, and it sometimes
leads to fatal syncope. This action on the left ventricle
by the influenzal toxin is probably the main cause of
the greater fatality of pneumonia when following an
attack of influenza, as well as of influenzal broncho-
pneumonia itself. In influenza the cardiac dullness
may extend one and a half or two fingerbreadths to
the left of the nipple-line; in a pneumococcal pneu-
monia it does not often extend much beyond the
nipple-line, and even this moderate increase seems
often to be caused by the distension of the right side
of the heart, for it may subside at once after a bleed-
ing. Thus it seems that the pneumococcus and its
toxin do not attack the ventricular wall so actively as
the influenza bacillus or the rheumatic diplococcus.

It must of course be remembered that the left
ventricle may be found enlarged as the result of
previous disease, rheumatic or otherwise, as the effect
of strain or of arterio-sclerosis or granular kidney. In
any case it adds gravity to the prognosis.

But the right side of the heart is in pneumonia
very soon overburdened by the pulmonary stagna-
tion, and it quickly shows signs of distress. Within
forty-eight hours from the onset evidence of dilatation
of the right auricle can often be detected by percus-
sion in the fourth right space. Unless means be
adopted to relieve it, the right side becomes increas-
ingly embarrassed as the days of the terrible pneu-

monia-week pass by. The normal fingerbreadth of dullness in the fourth right space becomes doubled, perhaps more than doubled, and dullness may be found in the third space as well. The first symptom caused by the distension of the auricle is an increase in the rate of the respiration. At a later stage this is accompanied by lividity of the lips and cheeks, with pallor. This lividity may increase to a general cyanosis due largely to the interference with oxygenation in the lungs. As the tension in the right heart increases, the patient feels much oppression and distress, and often becomes very restless, seeking ease in change of position and not finding it. This right-heart misery may be quickly relieved by a little loss of blood. In an early stage from three to six leeches will at once remove the distress, and the dullness of the right auricle rapidly diminishes to the normal fingerbreadth. The relief will usually last for forty-eight hours, after which time renewed distension of the right side may again demand relief. In a severe case it may be advisable to use leeches three times during the week but in many cases two applications of leeches will suffice to keep the right auricle from paralysing overdistension until the crisis

An old lady of 73, the mother of a Fellow of the Royal College of Surgeons, was seized with pneumonia of moderate severity. At first all went fairly well, but on the fifth day her pulse began to show intermissions, the lips became blue, the respiration-

rate rose to 40, and she became very restless. The
next morning (sixth day) she told me that she was
very ill and was going to die. I found that the right
auricle extended two fingerbreadths in the fourth
right space. Her son at once saw the necessity for
some removal of blood, in spite of her advanced age,
and eight leeches were applied over the lower right
ribs. In the evening she was again quite cheerful,
and I found that the dullness of the right auricle had
nearly returned to the normal fingerbreadth. Three
days later she again had a very restless night and
said that she was going to die. Again I found the
right auricle dilated. Three leeches only were enough
to give relief on this occasion. Next day she was
out of danger, and recovery followed. Some weeks
later I chanced to meet her in the street, when she
volunteered the remark, "You know, doctor, *those
blackies* did me good!"

By way of contrast, let me mention the case of a
much younger lady whom I saw in consultation and
found to be suffering from pneumonia. No attention
had been paid to the right heart; it was much dilated,
and the condition of the patient critical. Feeling sure
that it would be useless to propose a venesection, I
requested the practitioner in charge to apply leeches.
His reply was that he would "see if any could be
obtained in the neighbourhood." The patient died.

If the early leeching be omitted the right auricle
may become, as in this case, much distended, and

right-heart misery may be marked. In this condition it is better to have recourse to venesection and to remove 6 oz. or 8 oz. of blood. A larger quantity should be taken when the dilatation of the right heart is great, the pulmonary consolidation extensive, and moist *râles* heard over the rest of the lungs. In such circumstances 18 oz. or 20 oz. of blood must be taken if the patient is to have a chance of life. I have seen life saved even at this stage by a venesection to 18 oz. The blood obtained by venesection is often very dark in colour, and it sometimes spurts out as if from an artery when a vein is opened, showing the increased intravenous tension, and proving the need for relief of the intra-auricular tension.

In using the amount of dullness in the fourth right space as a guide when considering whether or not to bleed, it is necessary to be careful not to overestimate the size of the auricle, owing to pulmonary consolidation in its immediate neighbourhood or to pleural effusion, and, on the other hand, not to underestimate it through a local hyper-resonance of lung caused by compensatory hyper-distension of parts of lung not yet implicated in the inflammatory process. Of the two errors, probably the latter is the more serious, for it may tend to postpone a relief which the heart greatly needs. In case of doubt the practitioner will be wise to decide for the leeches ; they can hardly do any harm and they may do much good.

The total amount of blood removed by even three

applications of leeches or by a moderate venesection is really quite small. The plan here recommended is something very different from the excessive bleedings of former days, and the purpose is different. It is not with the idea of controlling an inflammatory process, but with the intention of giving relief to an overstrained right heart.

The repeated bleeding of cases of pneumonia advocated and practised sixty years ago was based on theoretical considerations. But it would hardly have been continued so persistently if some benefit had not been observed to follow its first employment in most cases. Doubtless, this benefit was the relief of the right heart unintentionally produced and the consequent diminution of dyspnœa and restlessness.

The most important part, then, of the treatment of pneumonia is *to keep a constant watch on the condition of the right heart, and to prevent its overdistension by the timely removal of a small quantity of blood.* This indication is, unfortunately, too generally neglected. The omission is probably responsible for a large part of the mortality from pneumonia. If a patient suffering from distension of the urinary bladder came under the care of a medical man, he would be at once relieved. But if it be the patient's right heart that is distended, a condition causing equal misery, equally dangerous, equally capable of being diagnosed, and equally easy of relief, the patient will probably be left to take his chance, because the practitioner's per-

cussion is inadequate and bleeding is out of fashion.
No improvement in the physical examination of
patients is more needed than an improved percussion
of the heart; no reform in therapeutics is more
urgent than a general adoption of the practice of
moderate blood-letting in some diseases of the heart
and of the lungs.

2. *Diet.*

It is obvious that the diet during an attack of
pneumonia must be such as can be taken without
effort, such as can be easily digested, and such as
shall readily be assimilated.   Milk fulfils all these
indications; it has also somewhat of a diuretic influ-
ence, and thereby will assist the elimination of toxin.

But there is one point which needs consideration.
The introduction of a fluid nutriment in large quantities
will tend to increase the quantity of fluid passing
through the right heart, and this is a matter of import-
ance when that organ is already overstrained.   For
the first two or three days of the disease milk alone,
in quantities of 3 or 4 pints daily for an adult, should
be given.   But when the right auricle is becoming
dilated it is wise to administer small quantities of a
highly concentrated and predigested nutriment.   A
useful resource at this time may be found in "malted
milk," a dry powder one-half of which consists of desic-
cated milk, the other half of malted wheat and malted
barley, with a little sodium and potassium bicarbonate.

Half an ounce of this dissolved in 2 oz. of milk may be given every hour while the patient is awake ; for a child the amount may be 2 teaspoonfuls in 1 oz. of milk. Thirst will probably not be felt for a day or two, and it may then be satisfied in another way, for as soon as the first leeches have relieved the right heart, it is possible to administer without harm considerable quantities of water. As much as 4 pints of water may be given in divided doses of half a pint every three hours during the day following the relief by leeches or venesection. This not only satisfies thirst but it helps to wash out the pneumococcal toxin, and thus is of great value. I have seen this measure produce a most marked effect in removing the delirium in a severe case of typhoid, and it has a similar influence in pneumonia.

3. *Sleep.*

In pneumonia sleep may be disturbed from the first by pain in the side, afterwards by continued pain, by dyspnœa and restlessness, by fever and rapid pulse, finally by cerebral congestion, and even by pneumococcal invasion of the meninges. Yet the refreshment of sleep is most important if the patient is to fight a winning battle, and the lack of it sadly diminishes his energy and power of resistance. *Every night's sleep is of importance.* Restless and disturbed nights in the early part of the attack are often regarded too lightly by the practitioner in attendance.

When the fourth or fifth comes, and the need for sleep
is urgent and distressing, he dares not administer
hypnotics because of the danger of inducing a fatal
coma. Whenever a medical man is called in at the
onset of an attack of pneumonia, let him never forget
to make sure that the patient sleeps during the first
three nights of his illness. Whatever other treatment
he may adopt, or refrain from adopting, let him take
measures to diminish pain and to secure sleep. If there
is pain which cannot be otherwise relieved, morphine
by injection under the skin is necessary, and at this
early stage is perfectly safe. If pain is slight or has
been relieved, a dose of bromide and chloralamide, or
of trional, or a Dover's powder, with hot brandy and
water, will suffice. But pain must be relieved and
sleep secured.

When the right heart is overstrained the procuring
of sleep is a difficult and anxious question. When
dyspnœa becomes distressing it is almost impossible
for the patient to sleep ; all his energies are needed
for the maintenance of the respiratory function. Mor-
phine given at this time diminishes the activity of the
respiratory centre, on the energy of which his life de-
pends, and may induce a coma which will end in death.
The way out of this dilemma is found in a relief of
the right heart.

After leeches or venesection the urgency of the
dyspnœa subsides, the cyanosis diminishes, comfort is
restored, and the wearied patient may fall asleep with-

out any narcotics.   This effect is often most marked
and most encouraging.   But, if necessary, a small dose
even of morphine may now be given with safety, or
other hypnotics if pain be absent.   The double effect
of the relief to the heart and the rest to the nervous
system gives the patient an enormous advantage in the
struggle which still lies before him.   Even at a late
stage of a severe case morphine may sometimes be
safely given, provided that the right heart has been
relieved by a venesection an hour or two previously.
But where a careful watch has been kept on the right
heart, and it has been duly relieved by leeches, and
at the same time a vigorous attack has been made
on the inflamed areas in the lungs by the free use
of ice externally, morphine is hardly ever necessary,
unless the pleura is severely involved and an empyema
is forming.

4. *Cardiac Tonics.*

Medicines which assist the heart to maintain the cir-
culation are often of much service in the later days of a
pneumonia.   It is generally advisable to begin the use
of these about the third or fourth day of the disease,
and they ought to be given, if at all, somewhat freely.

Of these drugs, strychnine is probably the most
useful.   It should be given by subcutaneous injection.
Half to one minim of the official solution twice daily
should be the initial dose for a child ; for an adult, 2
minims morning and evening at first, increasing to 5

minims if necessary. Even larger or more frequent doses may be given with advantage in influenza with dilatation of the left ventricle.

Atropine by subcutaneous injection is also very serviceable in children, but not so useful for adults, because they suffer much more than children from the dryness of throat and other unpleasant effects of belladonna. In children large doses of this drug will cause chiefly flushing of skin, which is of no importance. If the dose be further increased there may be dilatation of pupil, some mental excitement and restlessness, in some cases diarrhœa. The subcutaneous injection of 1 minim of the liquor atropinæ at first twice daily, afterwards every four hours, is often of the greatest possible service in the cardiac failure of diphtherial paralysis. I have used it for many years, and am convinced that it has often saved life. But it must be given freely, and one must not be afraid of a little delirium. Small doses are useless in a condition like this. Strychnine may be given with advantage at the same time, but I am sure that the atropine is the more useful. In the paroxysmal dyspnœa of mitral stenosis, with rapidly - increasing breathlessness, pallor, and sometimes evidence of dilatation of the right auricle, a subcutaneous injection of 4 minims of liquor atropinæ will often cut short the attack in a few minutes, and in children and adolescents will cause, as a rule, no unpleasant consequences. The same treatment is sometimes effectual in older patients, but in them the

after-results of belladonna are much more trouble-some.

In the pneumonia of children belladonna has been strongly recommended by Dr Coutts, and Dr Eustace Smith has told me of an apparently hopeless case of pneumonia in a child in which recovery followed the free use of belladonna, the child being delirious for three days. The tendency of the drug to cause delirium is a great disadvantage in using it for pneumonia, which itself is very prone to cause delirium. On the whole, it is a remedy much more useful for children than for adults.

Oxygen by inhalation assists the aëration of blood in the lungs, and thus improves the quality of the blood supplied to the cardiac muscle. It is, therefore, truly a cardiac tonic. Its use should be begun as soon as cyanosis is definite, and should be continued for five minutes every hour, whether the patient is awake or asleep. It can be given without disturbing him in the least. Oxygen is certainly a most valuable remedy, and ranks with strychnine in the treatment of pneumonia. But neither strychnine nor oxygen, nor both together, will often save life if the right auricle be not relieved. After a bleeding they are powerful remedies; without removal of blood they often fail, and almost necessarily. It is good to maintain the strength of the cardiac muscle; it is still better to diminish its labour. It is best to do both.

Digitalis will not always reduce the frequency of
the pulse in pneumonia, especially when the tempera-
ture is high.   It is most likely to be of service after
relief of the right heart, when the fever is moderate
and the pulse still remains weak and frequent.

Ammonium carbonate may be given when there
is evidence of much secretion in the bronchial tubes.

Alcohol, though called a " stimulant," has not much
title to be considered a cardiac tonic.   It is essentially
a vasomotor depressant, and as such may help the
heart indirectly when the tension is high.   There is
also sometimes a temporary increase in the strength
of the pulse after the administration of a moderate
dose, probably due to increased blood-supply to the
cardiac muscle, through relaxation of coronary arte-
rioles.   It is therefore possible that repeated small
doses may be of service in pneumonia, but the large
doses sometimes advised are likely to do more harm
than good.   To imagine that brandy can " support "
the heart when the right side is becoming paralysed
from overdistension is absurd.   In such a case the
only satisfactory cardiac tonic is a venesection.

5. *Ice.*

In pneumonia the application of ice to the wall of
the chest is as helpful as it is in pericarditis, but it
must be used much more freely.   A single icebag
will cover the whole anterior surface of the heart, but
the lung is a much larger organ, and for the efficient

treatment of an inflamed lung at least two icebags, often three, are required. If both lungs are attacked four icebags may be necessary in the case of an adult. It is, therefore, all the more important to keep in mind the two precautions which I have already mentioned : the feet and lower limbs must be kept continuously warm by means of hot-water bottles, and the right heart must not be permitted to be overdistended. The temperature should be taken and recorded every two hours ; in the case of a young child this should be done every hour. Of course the most efficient nursing is required and should be provided from the first.

The parts of the lung which are inflamed must be determined by careful percussion, and outlined on the chest with a blue pencil. The icebags must be carefully kept in contact with the skin over the areas thus determined, by passing their screw-tops through holes in a binder loosely fastened round the chest, or a vest may be cut out of domett as above advised in pericarditis. Special care must be taken that there is no leaking, also that they are surrounded by cotton-wool or a soft towel, and a pretty thick padding of such material is needed when the patient has to lie on the icebags applied to his back. This is really the greatest difficulty in the use of icebags in pneumonia ; the patient hardly ever dislikes the cold, but the pressure of the small lumps of ice on the back as the patient lies upon the bags sometimes

taxes the skill of the nurse to arrange matters com-
fortably.

At least once daily, preferably twice, the practitioner
must percuss the whole of both lungs as well as the
heart.   This must be done systematically and with
care.   Begin with the heart and determine the limit
of its dullness to the left, and especially its limit to
the right in the fourth and third intercostal spaces.
Notice the strength of the impulse of the left ventricle
and the force of the epigastric pulsation of the right
ventricle.   Listen to the quality of the first sound,
and observe the strength, rate, size, rhythm, and
length of the pulse-wave.   Notice whether the aortic
second sound, and especially the pulmonary second
sound, is too loud or too feeble.   Then percuss care-
fully the front of the lung and the upper axilla of
each side from above downwards quite down to the
anterior base, carefully noting and marking the limits
of any areas of dullness.   Listen for crepitant sounds,
especially inspiratory crepitation, and for bronchial
or tubular breathing, but do not fatigue the patient
by attempting to elicit bronchophony.   All this can
be done in a few minutes.   Then turn the patient
over on his right side and carefully examine the back
of the left lung from above downwards and the axillary
region, marking out all dull areas as before.   Let the
patient rest for a minute or two on his back, and
then turn him over on his left side and examine in a
similar manner his right lung and right axilla.   You

have now a fairly accurate idea of the extent and distribution of the areas of inflammation. In children these are almost always multiple and scattered over both lungs, some of the dull areas being partly due to collapse. But even in children the inflammation may tend to spread chiefly at one spot, and to involve a large part or even the whole of one lung, while the other is comparatively little affected. Yet, even in such so-called " lobar " cases careful percussion almost always yields areas of dullness in the opposite lung. And this is of great importance, for if these areas are allowed to spread, they may develop into such extensive areas of inflammation that the most careless percussor cannot fail to notice that he now has to deal with a " double pneumonia."

In adults the inflammation is usually more localised than in children, but there is the same tendency to spread. This extension of dullness can be easily watched by careful percussion practised daily, and a very good idea of the intensity and virulence of the inflammation may thus be obtained. This is requisite for the efficient use of the icebags. It is impossible to surround the whole of both lungs with ice ; it is therefore necessary to determine where the lung is inflamed, and in which direction the inflammation is spreading, and to apply the ice over precisely those areas. Place the first icebag on the largest dull portion of lung, and apply a second bag behind or in front, so as to enclose the inflamed area. These ice

applications are generally very pleasant to the patient.
An old lady of 62, after a few hours' trial of an icebag,
declared it to be "delicious."   A young lady of 19
said that it was "lovely," and when asked whether
she wished it continued, replied with emphasis, " Oh,
rather! yes!"   Nor is there usually any difficulty in
persuading the patients to try this method, for they
have generally already tried hot applications, and
have found little or no relief.   With a temperature
of 104° it is easy to understand that cooling means
comfort.   The difficulty is not with the patients, but
in overcoming the prejudices of the patients' friends.

At the next visit of the practitioner after the
application of the icebags let him remove them, and
carefully percuss again the areas to which they have
been applied.   He will generally find that they are
less dull than on the previous occasion, and that the
air enters them in inspiration better than before.   He
may even find that the dull area is somewhat smaller.
Encouraged by this observation he will replace the
icebags, and will proceed to attack any other dull
area that he can find by means of a third icebag.   At
the next visit he may find that the areas attacked
show signs of further improvement, or, at all events,
have not extended.   But the inflammation may at
the same time be spreading elsewhere ; new spots of
dullness may have appeared, or dull areas formerly
small may be rapidly extending.   This should be
suspected especially when the temperature continues

high in spite of the application of the ice, or when after falling it again rises. Then let the practitioner make diligent search for fresh and extending patches of dullness; he will often find them, perhaps in the lung he had thought unaffected. Care in this matter is of the greatest importance to the patient, for if such patches be detected in their earliest stage, it is remarkable how quickly they will yield to the local influence of the ice, but if they are overlooked or neglected they often increase rapidly, and may become extremely serious. It is specially desirable to have the ice over the spreading edges of the inflamed areas as far as this can be managed. It is often necessary to shift their position, more or less, at least twice daily, sometimes more frequently. It may be needful at every visit to give fresh directions to the nurse as to the exact areas to which the ice is to be applied. This is one reason why in pneumonia two visits daily are essential, three sometimes desirable. The progress of the conflict must be watched over the whole battlefield, during the whole struggle, by the general in command, if he is to secure the victory. The right heart and both lungs must be most carefully examined in detail at least once daily, a second time if the strength of the patient will allow it. It may sometimes be better to omit a minute examination at the evening visit, but at the morning visit the whole ground should be carefully gone over.

Thus far I have advocated the use of the icebag in

pneumonia solely on the ground of my own experience
of its practical utility.   But something may be said in
its favour from the standpoint of bacteriological
science.   For bacteriologists find that the pneumo-
coccus is remarkably susceptible to changes in its
environment, and in particular to changes of tempera-
ture.   Slight alterations in the composition of the
culture-media will affect its growth, so that it is very
difficult to cultivate.   Dr Eyre, who with the late Dr
Washbourn has specially studied this organism, tells
us* that it is "extremely susceptible to variations of
temperature."   He adds that "the range of tempera-
ture enjoyed by the pneumococcus is limited to about
14°, and is bounded by 28° C. on the one hand, below
which no growth takes place, and by 42° C. on the
other.   The optimum temperature is undoubtedly 37.5°
C."   It is, therefore, not unlikely that persistent local
reduction of temperature of a pneumonic lung, even
though of moderate amount, may exercise a definite
inhibitory influence on the rate of multiplication of
the developing pneumococci.   Fortunately this is
specially likely to be true in the worst cases of the
disease, for Dr Washbourn† showed from his experi-
ments that the greater the virulence the pneumococcus
is found to possess, the more marked is its susceptibility
to surrounding influences, and the more difficult it is
to cultivate.   Less virulent "strains" of this organism

* *Practitioner*, 1900, p. 285.
† Croonian Lectures, *Lancet*, 1900.

were able to grow at lower temperatures. He found that a pneumococcus which was slightly virulent, and which would grow on artificial media at a temperature as low as 20° C., could by passages through animals be converted into a highly virulent type, which would not grow on artificial media at temperatures below 37° C. If this is so, it suggests that the ice-treatment may have a special utility in the worst types of pneumonia.

It is difficult to estimate the amount of local reduction of temperature caused by the persistent application of ice over an inflamed lung, and there seem to be no available observations on the comparative rate of growth of the pneumococcus at temperatures differing from each other by 5° or 10°. I therefore requested Mr Neave, Bacteriologist to the Hospital for Sick Children, to make some investigations with regard to this. Mr Neave has kindly taken much interest in this research, and his report is as follows :—

" Some months ago, I undertook at your request to do what I could to test the cultivation of the pneumococcus at the temperature of the blood, and compare the growth with that at slightly lower temperatures. I confess the undertaking was made with a lighter heart than the exigencies of the matter really warranted, and that the results arrived at, to say the least, are far from complete. It would appear that each strain of the organism varies enormously in its idiosyncrasies of growth, and that a change is effected

after each cultivation on a medium which has been differently prepared in some minute particular. The great variety of statements as to the nature and behaviour of the pneumococcus, I feel sure, is due to these inherent differences. It is open to question what exact value can be put upon the planting of a number of organisms, and counting those that grow and make colonies. The late Dr Washbourn, in his Croonian Lectures, has shown that in cultivations that appear defunct, if a very large quantity is planted, one or two of surpassing vitality may yet be found to grow. I have, however, not been able to think of any better method of testing the effect of temperature on the growth of this organism.

"In the first place, I found that the isolation in pure culture of this organism was only occasionally possible, and that pleural-effusion strains, which were the most easily obtainable by me, had hardly ever sufficient vigour of growth on artificial media to enable a second or subculture to be made on a solid medium. This was necessary for the purpose of inoculating a plate with a definite quantity of liquid medium containing a growth of the organism. These difficulties I find well described in the *Baumgarten Jahresbericht*, 1900. However, by the kindness of Drs Klein and Gordon, an opportunity was given for the following :—A colony isolated from sputum was injected into mice, and the peritoneal and subcutaneous fluids resulting proved to be of exceptionally

vigorous growth.   In the first plating the peritoneal
fluid was diluted by sterile saline solution, and an
equal measured quantity was spread by a sterile glass
rod on two agar plates.   One was incubated at 39.4° C.
the other at 32° C., resulting in 285 colonies at the
higher temperature and 15 colonies at the lower.   It
would have been far more satisfactory to have used
the same liquid at the other temperatures required at
the same time, but there were only two incubators at
my disposal.   In consequence of this, a fresh cultiva-
tion had to be made every two days and used anew
for each subsequent comparison.   Thus no comparison
between one pair of groups and another can be made.
In the second comparison one plate was incubated at
37.5° C. and produced 1568 colonies ; while the second
at 27.5° C. produced 690 colonies.   In the third,
38.5° C. produced 153 colonies, and 32° C. produced
42 colonies.

   " The above, although consisting only of one set of
experiments, conclusively proves in respect of that
particular strain of the organism, that a temperature
of a few degrees less than that of the body prevents
so active a growth.

   " It may be that the comparatively more vigorous
growth at the lower temperature in the later experi-
ments, was due to the organism having been brought
up on artificial media, and so had become less virulent.
It may be noted that Dr Washbourn points out
that the non-virulent varieties have a stronger growth

at lower temperatures than those of a virulent character."

These observations appear to indicate that a moderate reduction of temperature does exercise a marked inhibitory influence on the rate of growth of the pneumococcus. If they are confirmed by subsequent and more extended investigations, they will furnish a scientific justification of the employment of the icebag in the treatment of pneumonia.

Whether or not this scientific basis can be claimed for the treatment, any one who will thoroughly and carefully employ it in the manner already detailed, and with the precautions above mentioned, will soon be convinced of its practical usefulness. Any estimate of its value founded on the use of one icebag only, in cases seen only two or three times in the week, and without any careful percussion of the right heart and any attempt to relieve it, is worthless.

One striking fact which ought not to escape notice is the great improvement which an efficient ice-treatment often produces in the physical signs before the crisis. This may be very distinct even two or three days before the crisis, and already very great indeed when it occurs. Another striking fact is the more rapid convalescence. This is natural enough, for if the microbic growth is inhibited there is less poisoning of the tissues, less dead material to be absorbed and eliminated, and less diminution of the patient's strength.

E

One point remains to be noticed. Some physicians look upon pneumonia as a blood-disease with local lesions, and would, therefore, consider the icebag treatment as irrelevant and useless. Now it is true that occasionally pneumococci have been detected in the blood of pneumonic patients, but Dr Washbourn held that it is only in a few severe cases that this can be done. This difficulty in detection indicates that the number in the blood is comparatively small. In whatever way the organisms may have reached the pulmonary alveoli in a case of pneumonia, whether by inhalation or by the blood current, it is certain that their chief, probably in most cases their only, seat of multiplication is the lungs. From the lungs they no doubt often pass into the pleura, sometimes into the pericardium, occasionally into the blood current, especially in the worst cases.

Dr Washbourn has proved experimentally that it is possible for pneumococci introduced into the air-passages to pass through the lungs, and produce pleurisy and pericarditis without any implication of the lung tissue itself. Clinically such cases are met with, but they are much rarer than pneumonia. Yet it remains true that the chief site of pneumococcal growth is in the air-cells of the lungs, and that the congestion and the production of fibrin are the local results of this growth and of the toxins thereby produced. On these points there is some valuable information in Dr Auld's *Seleeted Researches in*

Pathology, published in 1901.   He says : " If a pure, moderately virulent culture of the pneumococcus be inoculated, either subcutaneously or into the internal cavities of a susceptible animal, it produces around the site of inoculation intense congestion, exudation of sanguineous serum, and fibrin.   That is its characteristic and invariable lesion, and it produces directly no other.   Should resolution not occur soon, we have, of course, after a variable time, an exudation of leucocytes, the gradual collection of which may give rise ultimately to what has the appearance of a mass of pus."

After inoculating beneath the skin of a rabbit rusty sputum from a case of pneumonia, and killing the animal within thirty-six hours, Dr Auld found that its heart-blood contained a pure culture of the pneumo-coccus.   He grew these on solid and liquid media, and made injections into the pleural cavity of rabbits, causing always a severe pleurisy, frequently severe congestion of the lungs, often more or less consolida-tion, in two cases pneumonic consolidation of an entire lobe, usually also pericarditis.   The local lesions, lungs, and spleen of these rabbits were subjected to a chemical analysis, and from them were obtained an albumose and an organic acid.   The latter gave the reactions for lactic acid ; it was not toxic on injection into animals.   Dr Auld suggests that the production of this acid may have an inhibitory effect on the further growth of the pneumococcus.

But the albumose acted very differently. When injected beneath the skin of the rabbit's ear, local inflammation of the ear was produced and a rise of temperature ; recovery followed. Intravenous injection produced initial shock, followed by a marked rise in temperature, but no other pathological effect. Injection into the pleural cavity produced no initial depression, but marked dyspnœa soon set in, followed by rise of temperature. The animal was killed on the third day, and found to have pleurisy on the right side, with complete consolidation of the lower lobe of the right lung. No other lesions were found. Inoculations were made from the blood, and also from the serum in the pleural cavities, in solid and liquid media, with a negative result. A larger quantity of the albumose was injected into the pleural cavity of another rabbit. Next day the temperature was 104.6°. On the following day the animal was killed, and severe right lateral and also diaphragmatic pleurisy was found, with pericarditis and hard consolidation of the upper part of the lower lobe of the right lung, very typical of ordinary lobar hepatization.

Thus it seems that the consolidation of lung in pneumonia is largely due to the local effects of an albumose produced by the growth of the pneumococcus, and that the pyrexia and probably also the tendency to delirium, are due to its action on the brain.

A similar albumose was obtained, in smaller

quantity, by growing the pneumococcus in albuminous media free from albumoses, and it was found to produce similar results on injection into rabbits, thus proving that the pneumococcus has the power of forming a highly toxic albumose from proteid material. Much larger doses, however, were required, showing that the production of toxin is much more active in the living body than in artificial media.

Another interesting observation of Dr Auld's may also be mentioned. He found, by injection of pneumonic toxins obtained by filtration of cultures of the pneumococcus, that the effect of such injections was to make the animals subjected to them more susceptible to subsequent infection with the pneumococcus than normal animals. This susceptibility appeared to last for a long period. The clinical importance of this fact is obvious.

LECTURE III

Pneumonia.

IT may be well to combine the suggestions of the preceding lecture into an outline plan of treatment for pneumonia, though it may involve some repetition.

Every case in which a rigor occurs and the temperature rises should be sent to bed at once in a well-ventilated room without draughts, the warmth of the room being maintained at 60° F. If pneumonia is apparently developing, a trained nurse should be obtained from the first. The temperature, pulse-rate, and respiration-rate should be observed, and recorded on a chart, and this should be repeated every four hours. If the patient, when first seen, is cold and at all collapsed, it is desirable to give him a " hot pack," by swathing him in a sheet wrung out of hot water of temperature of 110° F. (the head being kept cool), and covering him with blankets. Some hot brandy and water may be given to him to drink. He should be kept in the pack for about twenty minutes, then

the sheet should be removed, the patient dried quickly,
and placed in a warmed bed.

When he has thus been rendered warm, let the
practitioner make a careful examination of (1) the
tongue, mouth, throat, glands, (2) the left heart, (3)
the right heart, (4) anterior pulmonary regions, (5)
posterior and lateral pulmonary regions, (6) liver,
spleen, abdomen. If pneumonia is developing, it is
usually possible, by a very careful percussion, to
detect some slight indication of the coming trouble,
and it is extremely important to make sure of the
diagnosis as soon as possible, for before the expira-
tion of twenty-four hours from the onset there is a
chance of arresting the disease by vigorous treatment.
There will probably be pain on one side of the chest,
with somewhat limited expansion of that side in inspir-
ation, and some slight local impairment of resonance
at base or apex. Over this area there may be a very
little subcrepitant *râle*, but the chief auscultatory
indication will be local feebleness of breath sounds.
This comparative absence of breathing in the earliest
stage of pneumonia is mentioned by Professor Osler,
but is not generally recognised : it is certainly a fact.

Put two hot-water bottles to the patient's feet, and,
as soon as possible (every hour is of importance), fill
two icebags with small fragments of ice, and apply
them as already directed over the suspected part of
the lung, one in front and one behind. If the mouth
and fauces are foul, a sanitas mouth-wash should be

employed, and the throat sprayed with perchloride of
mercury lotion (1 in 2000).   This should be repeated
every three hours for the first two days.   It is prob-
ably desirable in every case, for the infection of the
air-passages doubtless often starts from the mouth,
and the spraying can be easily effected during the
early days of the attack, when there is little dyspnœa.
The diet should consist of milk, or milk and barley
water, given every two hours, and water if desired.

The patient should be seen again the same evening
and again carefully examined.   Any other area of
dullness that can be detected should be covered by a
third icebag.   If pain in the side has not been already
relieved by the ice, a subcutaneous injection of $\frac{1}{6}$ gr.
to $\frac{1}{4}$ gr. of morphine should be administered, and a
night draught of bromide and chloralamide should be
ready if the patient does not sleep.   This must on no
account be overlooked.

If the attempt at arrest is successful, on the second
day the dullness will be found not to have increased
—possibly it may already have diminished ; the air
will enter the suspected area more freely, the tempera-
ture will be lower, and the pulse-rate less frequent.
It will in this case be necessary simply to persevere
steadily with the treatment, but the greatest care
must be employed to detect any fresh inflammatory
foci, and to attack them immediately.   Carelessness
in percussion will lose the possible chance of saving
the patient from a dangerous illness.

In proof of the assertion that if a case of pneumonia comes under observation within twenty-four hours after the initial rigor, it is sometimes possible to arrest it by vigorous treatment, I give the two following cases :—

CASE I.—W. B., 18, carman, was seized on the evening of 31st October 1895, sixteen hours before his admission into St Mary's Hospital, with a rigor which lasted an hour. Next morning he had fever and pain in the right side. On admission his skin was hot and dry, and there was some labial herpes. Temperature, 103.6°; pulse, 120; respirations, 40. When I first saw him on the evening of 1st November, twenty-four hours after the rigor, I found dullness at the base of the right lung in front below the fourth rib, extending into the lower axilla, with some tenderness. The breath-sounds were feeble over the dull area. No bronchial breathing, but a little crepitation at the end of inspiration. Behind, at the right base, breathing weak, and some impairment of resonance. Three icebags were at once applied.

2nd November.—Temperature, 101.8°; pulse, 100; respirations, 34. Feels better. Dullness decidedly less extensive.

3rd November. — Temperature 100°, rising to 101.8°, falling to 99°; pulse, 100; respirations, 30. Dullness still diminishing. Says he is " a lot better."

4th November. — Temperature, 98°; pulse, 72; respirations, 26. Now only a small dull area in lower axilla. Ice removed (after sixty hours).

5th November.—Temperature rose to 99.8°, but fell to 98°.

6th November.—Temperature rose to 100.2°, but fell to 98°.

7th November.—Temperature, normal; pulse, 64;

respirations, 20. Very slight impairment of reson-
ance could now be detected.

CASE II.—E. N., 14, admitted 22nd May 1896,
twenty-four hours after immersion in a canal and
twelve hours after a rigor. He had a headache and
dyspnœa. On admission, dullness was found in the
right axillary region, and an icebag at once applied.
Temperature, 103°; pulse, 120; respirations, 40.

23rd May (10 A.M.).—Both cheeks very flushed.
Obvious dyspnœa. Temperature, 103°; pulse, 120;
respirations, 40. Dull in the right lower axilla, not
behind scapular line nor to inner side of nipple. Just
below angle of right scapula there was distinct fine
crepitation, with inspiration only; this was so typical
that I made all my clinical clerks listen to it. Breath
sounds diminished over the dull area; no bronchial
breathing. Dullness and diminished breathing in
right suprascapular fossa also. Some pain on left
side of abdomen on taking a deep breath, but no rub
could be heard and there was no dullness. Heart
normal. Three more icebags were ordered, making
four in all; two to the right base, a third over the
right apex behind, and the fourth over the left axilla.
After one hour the temperature fell to 100°, and the
ice was removed. It then rose to 102°, but at once
fell again.

24th May—(forty-eight hours after the rigor).
Temperature, normal; pulse, 74; respirations, 34.
He had slept well, was now not flushed, and the right
axilla was less dull. The temperature remained sub-
normal for thirty-six hours. There was a short rise
to 100° on the 25th, and to 99.5° on the 26th. After
this it was normal, and the boy was quite well, and
the right axillary region was of normal resonance.

It will be observed that in each of these cases there

was no crisis, but an immediate and rapid subsidence of temperature, physical signs, and symptoms.   In such cases as these it is reasonable to claim that the disease has been arrested. But it is not always possible to arrest a pneumonia even when it is treated very early, and after twenty-four hours there is little hope of success. This is not surprising when we remember how rapidly micro-organisms increase in number under favourable circumstances.   Washbourn and Eyre found, on cultivating the pneumococcus in nutrient broth, making plate-cultivations from the broth-culture and counting the living cocci present at different periods, that 140 colonies increased in three hours to 6149, and in six hours more to 13,680 ; twelve hours later they were " innumerable."

As the normal temperature of the human body is only 1° F. below the optimum temperature for the growth of the pneumococcus, it is clear that if an attempt to arrest the development of a pneumonia is to have any chance of success, it must be made very early and very vigorously.

But it is always possible to influence the course of a pneumonia, to diminish its intensity, and often to shorten its duration. This of course is difficult to prove, because of the uncertainty of the time of occurrence of the crisis in the disease when untreated. But there is nothing really improbable in the assertion that there is reason to believe that the ice-treatment sometimes brings about an earlier crisis. For how-

ever the crisis may be caused, whether by the manu-
facture of an antitoxin or by a failure of further
growth of the pneumococcus, it seems clear that any
treatment which can to any extent inhibit the growth
of the microbe and thus check the amount of toxin
which it produces, will to that extent facilitate the
earlier termination of the struggle between the
attacking and the defending forces ; in other words, it
will hasten the crisis.

If the attempt to arrest the disease is unsuccessful,
on the second day the area of dullness will be larger,
and over it may be heard inspiratory crepitation, or
sharp *râles* of double rhythm in children, or some
prolonged expiration, or distinctly bronchial breath-
ing. A third or a fourth icebag should now be
applied, the sites for their application being outlined
in blue.

It is desirable at this period to administer 2 or
3 grains of calomel, followed after three hours by
a seidlitz powder. When a sufficient evacuation has
been obtained the purgative should not be repeated,
for in the later days of a pneumonia there is a
tendency to diarrhœa.

On the second evening the hypnotic must be given
again if necessary, and morphine if pain is present,
for the patient must have sleep. It may, perhaps, be
desirable to remove one or two of the icebags during
the night, leaving two only in position. It might be
thought that the necessary disturbance would be

fatal to sleep, but the relief of pain and dyspnœa is so great that the patient easily falls asleep again, provided that his right heart is not over-full.   In the case of young children the temperature should now be taken every two hours (hourly for babies), and it can be done without disturbing them.   If any icebags have been removed at night, they should be replaced early next morning.

On the third morning the physical signs in the lungs must again be most carefully determined, and directions given for the alterations of position of the icebags necessitated by the changes found.   But now special attention must be given to the right heart. If the dullness of the right auricle is found to extend two fingerbreadths in the fourth right space, and there is distinct dyspnœa and some slight lividity of lips, or cheeks, or finger-tips, leeches should be applied over the lower ribs on the right side below the nipple-level.   One should be used for a baby under six months, two for a child under two years, four for a child of ten years, six for an adult, eight for a robust man.   If not used at once, the leeches should be held in readiness, for they may possibly be required in the evening if the patient is to sleep. Some "malted milk," and one or two cylinders of compressed oxygen should be procured.

On the third evening it will in most cases, unless the ice has already caused a marked improvement in the physical signs, be advisable to apply

leeches—if they have not been already used—an hour
or two before the time for sleep. The relief thus
given to the right heart will often induce sleep with-
out any hypnotic, but one must be given if needful.
Even morphine may be used safely under these
circumstances.

On the fourth morning, if the leeches have been
applied, the patient will feel more comfortable, though
the physical signs may have increased in extent.
The same minute care in determining the physical
signs in both lungs must be practised. Watch care-
fully for fresh areas of dullness, especially if there has
been any fresh rise of temperature, and attack them
at once.

The right auricle having now been relieved, it will
be desirable to give considerable quantities of water,
both to satisfy thirst, and to promote diuresis and the
elimination of toxin. During the twenty-four hours
following the use of the leeches, 3 or 4 pints of water
may be given, in quantities of 8 to 10 oz. every three
hours ; for a child, 4 to 6 oz.

If the patient has not come under treatment until
the fourth day of a severe attack, he will probably be
in considerable distress. Dyspnœa and discomfort
will be marked, cyanosis distinct, and the dullness of
the right auricle may measure from two to two and a
half fingerbreadths in the fourth space, one or one and
a half in the third, and half a fingerbreadth or more
in the second. This should be ascertained at once,

before any attempt is made to discover the amount of disease in the lungs. The call for bleeding is urgent and imperative. A larger number of leeches must now be used than would have sufficed on the previous day. Two must be employed for a baby, three for a young child, four to six for an older child, eight to twelve for an adult. A venesection is often preferable : 4 oz. for a young adult, 8 oz. for a strong man.

An hour after the bleeding both lungs should be carefully examined and the outlines of the dull areas marked on the chest. Two icebags must be applied at once to the worst inflammatory foci, an hour or two later a third, and before long a fourth. We are now in the thick of the fight, and it is necessary to call up the reserves and have all our forces in readiness for the struggle of the next three or four days. The subcutaneous injection of strychnine should be begun and maintained systematically in increasing amount or frequency. Now is the time also to begin the administration of oxygen ; this, too, should be regularly continued throughout. Milk and also water may be given in considerable quantities after the venesection. At night sleep will probably come naturally, the right heart having been relieved, and the pulmonary congestion diminished by the ice, but if not a hypnotic must be given, and even morphine if necessary ; the patient must have sleep.

On the fifth day, if the patient has been vigorously treated with leeches and ice, there is often a marked

improvement in the physical signs, and much less
tendency to extension.   But, if this be not the case,
it will now be desirable to limit the amount of
fluid given to the patient, so as to lessen the
strain on the right heart.   The diet for the next
two or three days may be simply malted-milk
powder dissolved in milk, a tablespoonful in 2
oz. hourly for an adult, two teaspoonfuls in 1 oz. for
a child, while awake.   The icebags must be con-
tinuously applied, and their position altered as may
be necessary, special care being taken to discover and
attack fresh or spreading areas of inflammation.   If
leeches have been used on the third day, it is desirable
to examine the right heart again very carefully on
the evening of the fifth day.   The relief will almost
always last for forty-eight hours, but by the fifth
evening some more leeches may be required.   In
determining this point, especially when the left lung
is mainly involved, it is very necessary to guard
against being misled by overdistension of the right
lung into an underestimate of the size of the right
auricle.   In case of doubt, let the decision be for the
leeches.   The amount of sleep which the patient has
hitherto obtained is also of importance in deciding
this question.   If he has slept well, and the right
auricle does not measure more than two finger-
breadths, the leeches may be postponed.   But if
sleep has been defective, it will be wiser to apply
them, and afterwards to give a hypnotic.   For sleep

is of great importance for the maintaining of vigour
for the days which may remain.

If the patient has reached the fifth or sixth day of
his illness, and neither blood-letting nor ice has been
employed, the symptoms are often very severe, the
distress great, and the outlook gloomy. Probably he
has been sleepless for several nights, and his strength
is rapidly diminishing. The call for active treatment
is urgent. The prognosis depends on three factors :
the age and previous health of the patient, the
intensity of the infection, and the action of the medical
attendant. At such a time the responsibility of the
latter is great indeed. Life is trembling in the
balance. His action or his inaction may decide
whether or not the patient shall be deprived of many
years of life, and his wife and children suffer an
irreparable loss.

The first necessity is a venesection. Eight ounces
of blood should be taken at once, twice as much or
more if the lung be full of *râles*. If permission for
venesection cannot be obtained, place a dozen leeches
over the liver and encourage the bleeding. Hypo-
dermic injections of strychnine in 3-minim doses every
four hours should follow immediately, and the syste-
matic inhalation of oxygen for ten minutes or more
every hour. Two icebags should be at once applied,
soon followed by a third, and before long by a fourth.

It is very probable that after this treatment the
patient will fall asleep. If so, he should be undis-

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turbed for four or six hours.   But after this time
nourishment must be given and the icebags refilled
every two hours.   Malted-milk in milk with a little
brandy should be given every hour when he awakes,
and all medicine by the mouth avoided.   After sleep
has been obtained a small enema may be given if
necessary.   If diarrhœa is present, the rectum should
be washed out with warm saline solution, and 2 oz.
of starch decoction with a few drops of tincture of
opium inserted.

Some improvement—often much—will certainly
follow this treatment unless the patient be already
very far on the downward road, or his heart be
previously dilated, his lungs emphysematous, his liver
cirrhosed, or his kidneys granular.   Many cases are
no doubt hopeless from the first ; but not very rarely
an apparently hopeless case recovers ; and, at all
events, whatever can be done to give a chance of
recovery ought to be done.   Here let me put in a
plea for earlier consultations.   Too often a " second
opinion " is sought for only when death is imminent.
The surgeon is right in asking that he may be
allowed to see a case of perforated gastric ulcer as
soon as the diagnosis is made ; if twenty-four hours
are allowed to elapse, the patient's chance of recovery
is small indeed.   It is estimated by Mr Mayo
Robson that if operated on within twelve hours after
the perforation the mortality is only 16.6 per cent. ; if
within twenty-four hours, it is 63.0 per cent. ; if

within thirty-six hours, it is 87.5 per cent. ; and if
delayed for forty-eight hours, the operation will only
rarely succeed.   So may the physician plead that in
pneumonia the final issue largely depends on the
treatment, or want of treatment, during the first few
days.   In the case of an infant, or of an adult older
than 30 years, the danger to life is great, and judicious
treatment is required from the very first.   This is not
so obvious to the patient as when an operation is
required, but it ought to be equally obvious to the
practitioner.   To delay the consultation in such a
case to the fourth or fifth day is to imperil the
patient's life.

Pneumonia in adults usually ends by a very rapid
fall of temperature, with slowing of pulse.   This
" crisis " often occurs in children also, even in cases
which would be designated as " bronchopneumonia,"
but in children the subsidence is apt to be more
gradual, and to occupy several days.   Pneumonia in
children sometimes lasts for three or even four weeks.
It is necessary to keep a careful watch for the first
indications of this quick diminution of temperature,
especially in children.   The icebags should be gradu-
ally removed as the temperature falls, and the last
should be taken off when the thermometer marks
100° F.   There is a natural tendency to collapse at
the time of the crisis which must be kept in mind.
Icebags over the chest at this stage would probably
be injurious, though in pericarditis, as I have already

mentioned, they may sometimes be used with advantage, even when the temperature is subnormal. If, in spite of care, or for want of it, the reduction of temperature is so great as to cause some collapse, it is desirable to apply warmth over the heart and abdomen, also to the feet, and to give the patient some hot water with brandy, and a draught containing ether and ammonia. By these means it is usually easy to remove any tendency to collapse.

But the crisis is often preceded by a remission of temperature which lasts only a few hours. Hence, when the ice has been removed, the temperature should still be taken every hour. If it rises to 101° F. a single icebag should be again applied, if to 103° F. at least two. Much careful observation on the part of the nurse is required at this period. If the temperature shows a persistent tendency to keep above normal after the crisis has occurred, the existence of empyema should be suspected, and an exploring needle should be passed into the dullest area. Occasionally it may be due to tuberculosis.

Empyema.

The frequent occurrence of empyema as a chronic condition following an attack of pneumonia is well recognised, but I wish to call attention to what may be termed acute empyema, in which the pneumococcal invasion mainly or exclusively involves the pleura, the lung being little affected, perhaps not at all. This

seems to be more common in children, but it may occur
in adults.   In a young child in the Hospital for Sick
Children, who had a temperature of 106° F. and
dullness over one base, and who was thought to be
suffering from pneumonia, the necropsy revealed a
considerable amount of turbid fluid in the pleural
cavity, but no consolidation of lung.   In a girl
recently under my care at St Mary's Hospital, 3 oz.
of pus were obtained from the left pleura as early as
the eighth day of an attack which was at first
thought to be merely pneumonia, but in which the
extensive dullness, feeble breath-sounds, and cardiac
displacement soon suggested pleural effusion. I think
that in this case some pneumonia was present also, for
cyanosis was well marked, and the dullness of the right
auricle in the fourth right space measured two and a
half fingerbreadths on the third day of the illness.

When the amount of pus in the pleura is small and
localised by adhesions, it is often extremely difficult
of diagnosis.   Probably few physicians have escaped
the mortifying experience of the *post-mortem* dis-
covery of an empyema which had been overlooked, or
diagnosed as some other condition.   The difficulty of
diagnosis is similar to, though not quite so great as,
the difficulty in suppurative pericarditis, and the two
conditions are sometimes associated.   There may
be a small amount of pus in both pleuræ as well as
in the pericardium ; it is rare to find any in the
peritoneum.

A localised empyema may be diagnosed as a pneumonia, as a serous pleural effusion, as a bronchiectasis, as tuberculous consolidation of lung, as collapse of lung, or as a subphrenic abscess, and the diagnosis may be impossible without the exploring needle. The signs of distinction between a consolidated lung and a pleural effusion may be neatly stated and tabulated in text-books, but there is not one of them which may not prove fallacious in practice. The temperature may be high in an acute empyema ; it may be low in pneumonia. The breath-sounds are sometimes audible in children over a pleural effusion, and they may be quite inaudible in an influenzal bronchopneumonia, or in the later stages of a lobar pneumonia. Even displacement of the heart is not certain proof of a pleural effusion, at all events in children ; it may sometimes be caused in them by a solid lung. This is especially true of a form of tuberculous disease of the lung in which the lower and middle lobes on the right side are attacked by tubercle spreading from the bronchial glands. This occurrence is not very rare in children, and my colleague, Dr Batten, has shown from *post-mortem* observations that it is much more frequent on the right side than on the left. Clinically this condition may cause all the physical signs of a pleural effusion, including some displacement of the heart, as I have seen in a case in which an incision revealed a healthy pleura, with solid lung beneath. In some cases a

small localised empyema between the lobes of the lung may coexist, as in a girl under my care at Great Ormond Street. In this case Mr Collier succeeded in draining the small, deep-seated cavity, and the patient gradually recovered.

The danger of overlooking an empyema which is small, or even of moderate size, is so great that it should always be kept in mind, and an exploring needle should be passed if there is any room for doubt. If the first puncture does not yield pus, an anæsthetic should be given, and punctures made in other sites of dullness. If pus is found, an incision should at once be made, a piece of rib removed, and a drainage tube inserted.

Pleurisy.

Acute pleurisy in its earliest stages may be very successfully treated with ice to the chest. One icebag is often sufficient ; it should be applied over the spot where friction is audible. It quickly relieves pain, and often renders morphine unnecessary, though it might be wise to give a small amount of this drug hypo-dermically if the pain were very severe. Two icebags may be used if necessary. The friction often rapidly disappears after the ice has been applied for some hours, and the tendency to effusion is very decidedly checked. As an illustration of this beneficial effect, I may mention a recent case of dermoid cyst of the right thorax under my care at St Mary's Hospital :

A large cyst displaced the heart into the left axilla and compressed the right lung. By four distinct operations, at considerable intervals, Mr Silcock succeeded in removing the whole of the wall of the cyst, dissecting it off the diaphragm, the right pleura, and the pericardium. On the day following the last of these operations the patient complained of pain in the left side, and the temperature rose. Over the cardiac region and to the left of it a fine crepitant friction could be heard, with the respiration only. An icebag was applied. Next day there was a definite pleural rub in the left axilla, and pain continued, though less intense.

On the day following the pain was much relieved and the rub less marked, but there was much inspiratory dyspnœa, both sternomastoids acting strongly with each inspiration. It was impossible to determine accurately the extent of the dullness of the right auricle, owing to the previous condition and to the operation, but it was clear that it was greatly overstrained through the occurrence of pleurisy and probably acute pulmonary congestion in the single lung that was able to perform the respiratory process. We were very reluctant to submit the patient to any further loss of blood, and fortunately we succeeded by a subcutaneous injection of morphine and atropine in escaping this necessity. Next day the danger was past, and the patient steadily recovered.

I think it is practically certain that but for the icebag this patient would have died.

In the treatment of a large serous effusion ice externally is very useful, but it should be preceded by paracentesis. As soon as it is evident that there is a considerable amount of fluid in the pleura, it is

best to aspirate at once, under low pressure, and draw off as much fluid as comes easily. Then fix this side of the chest with broad bands of strapping firmly applied, and over the strapping apply two, or three, icebags. This treatment rapidly relieves the discomfort and dyspnœa, and any pain that may be present. If the ice is maintained in position for about two days there is usually little tendency to reaccumulation of fluid. But if a relapse does occur, it may be treated in the same way, and the ice applied for a longer period. The serous fluid obtained by paracentesis in such cases is usually sterile ; it is probably often due to tuberculosis affecting the pleura. It is frequently possible to obtain physical evidence of this by a careful examination of the *opposite* lung, for there is in many such cases more or less dullness and feebleness of breath sounds at the inner end of the first space and over the posterior part of the upper apex, also below the apex of the lower lobe. This is the more striking because the rest of this lung is often hyper-resonant.

In an acute local pulmonary tuberculosis icebags applied over the dull apices often give much comfort, and appear sometimes to be of distinct service in checking the rapid progress of the disease. This is especially the case when the temperature is high.

In acute laryngitis an icebag placed over the larynx gives rapid relief to the symptoms, and in a catarrhal laryngitis is curative.

In local peritonitis over gastric ulcer, with threatening perforation, ice over the epigastrium brings much comfort to the patient, and greatly aids the subsidence of symptoms.

Appendicitis.

In the treatment of inflammations of the appendix vermiformis the persistent application of an icebag is far more effective than fomentations or poultices; it rapidly relieves pain, and obviously diminishes the local inflammation. Clinically, cases of appendix-inflammation may be divided into three groups. The first and most important group consists of cases of perforation of the appendix, with escape of concretions or of pus into the peritoneal cavity, or of a gangrenous condition of the appendix itself. Such cases are usually of sudden onset, and the symptoms often severe. Pain, vomiting, tenderness, rigidity of abdominal muscles, flexion of the thigh, limitation of descent of diaphragm during inspiration, tympanites but often no local tumour, with evidence of septic absorption, are the chief indications. Such cases demand immediate operative interference; every hour is of importance. They are similar to cases of perforated gastric ulcer, or to a strangulated hernia. No question of palliative treatment must be entertained for a moment. The absorption of septic material from the peritoneum is often very rapid, and a delay of a few hours may make

all the difference between life and death. No such case ought to be allowed to die without operation; not seldom, apparently desperate cases have been rescued. The operation ought never to be delayed because of the collapsed condition of the patient; it is the most effectual means of removing the collapse—often the only chance.

The cases of the second group are much more common. They are often due to a subacute inflammation of the appendix, which may be thickened. The symptoms are comparatively slight, but there is usually some definite local tenderness, and often a tender local swelling can be detected, with dullness on percussion over it. This swelling may sometimes be due to a local muscular contraction in the abdominal wall, as suggested by Dr Mackenzie, but it certainly appears in some cases to be the thickened appendix itself which is felt.

These cases will usually recover in time under any or under no treatment, but they are prone to relapse. Fomentations or poultices give some relief to discomfort, but they do not cause rapid subsidence of the swelling and of the symptoms. This, however, is often promptly effected by an icebag, the results produced by which are sometimes very striking.

Intermediate between these two groups of cases, clinically speaking, is a third group, in which the symptoms are very definite, but not so acute as to make it clear that immediate operation is required.

There is usually a distinct local fullness and tender-
ness. The practitioner is in doubt whether pus has
already formed, and whether an incision ought to be
made at once or delayed. In this difficulty, which is
by no means rare, and in which a correct decision is
of the greatest importance, I have found that the
application of an icebag for three or four hours will
often give the necessary guidance. If after four hours
the ice has not given distinct relief, an operation
should be performed without further delay. But if
there be definite, even though slight, alleviation of
symptoms, the operation may be postponed for a
short time and further trial made of the icebag. In
many cases after twenty-four hours the relief is so
definite that no further question of immediate opera-
tion need be entertained. If the improvement con-
tinue, the icebag should be kept in place persistently
for two or three weeks, with absolute rest in bed.
Such cases must be very carefully watched, for a
small local abscess may remain and may require
incision at a later period. But the condition will
then be much more favourable for operation than at
first, the pus will be well localised, and the surround-
ing inflammation quieted down. On the other
hand, if the early relief is not maintained, although
the icebag has been applied persistently, it will
usually be wise not to delay the operation any longer.
Thus the icebag becomes a useful test by which we
may decide the often difficult and anxious question

as to whether or not an operation ought to be per-
formed.

If recovery has occurred without operation, it must
be decided whether or not the appendix should be
removed.   If this is the first occasion on which it has
offended, and the symptoms have been slight, it may
surely be allowed another chance, though this is not
the opinion of the most "advanced" surgeons.   But
if the symptoms have been severe, or if it be a second
offence, probably removal after a short period of con-
valescence is desirable.

Acute Nephritis.

The last form of acute visceral inflammation of
which I desire to speak is acute nephritis.   The
method of treatment usually advised for this serious
malady is often very unsatisfactory in its results.
Recovery is slow, and in a large number of cases
imperfect.   In the worst cases, if death from acute
suppression or rapid uræmia does not supervene,
the patient passes into a condition of chronic albu-
minuria and dropsy, becoming, as my former teacher
the late Dr Moxon expressed it, "a large white man
with a large white kidney."   In the majority of cases
more or less albuminuria remains after apparent
recovery, and in many there is relapse of inflamma-
tion in the damaged organs, leading directly or in-
directly to a fatal issue.

Even the etiology of acute nephritis is very

imperfectly understood. Scarlet fever is, of course, responsible for a very severe form of nephritis. Diphtheria often causes transitory albuminuria, more rarely a real nephritis. The same may be said of influenza, typhoid fever, and pneumonia. The rheumatic diplococcus has been found in the urine of rheumatic patients, and it must, in the future, be a subject for inquiry whether an attack of acute rheumatism may possibly be responsible for slow insidious changes in the kidney, whether, for instance, it may be one of the causes of granular kidney. The "cold and wet," to which some cases of acute nephritis are ascribed, probably act, as they do in the production of pneumonia, by permitting the internal cultivation of parasitic micro-organisms, and the elimination of their toxins. Many cases of subacute nephritis in children and young adults are difficult of explanation ; in many of them there has been no ascertainable exposure to cold and wet or to infection from scarlet fever. Perhaps some may be due to the injury done to the kidneys by microbes or microbic toxins from the alimentary canal eliminated in the urine. In the worst case of nephritis that I have seen in a very young child (aged $2\frac{1}{2}$ years), the only fact that could be ascertained which seemed to offer any explanation was that he had suffered from an attack of vomiting and diarrhœa which lasted about a fortnight. Soon after his recovery from this, about three weeks from its onset, he was noticed to be "growing fat." But this "fat" was the dropsy

of an acute nephritis, and his urine was solid with albumen. He was treated in the manner usually advised, and after a long and trying illness gradually made a very imperfect recovery. Considerable albuminuria remained when he was discharged from the hospital. This result is certainly not a therapeutic victory. Can we do nothing better for such patients? I think we can. The external application of icebags over the inflamed kidneys in nephritis has, in several cases, given me excellent results. I have less experience of the use of ice in nephritis than in pericarditis or in pneumonia, partly because the cases are less common, but mainly because of the apprehension, only gradually overcome, that the attempt might result in a fatal suppression of urine. But in this case also time has proved to me the wisdom of the old adage, " Don't think ; *try*." For it has turned out that when used with proper precautions ice over inflamed kidneys, far from causing suppression of urine, is an excellent diuretic, rapidly increasing the amount of urine passed. No doubt there might be danger of inducing suppression if the ice were applied while the skin was dry and cold, and the patient more or less collapsed. For indeed suppression may be caused by the disease itself, as I found recently in the case of a woman, aged 36, whom I saw in consultation a few hours before her death on the sixth day of an acute suppression. A catheter passed into the bladder removed about an ounce of turbid urine full of

albumen. No treatment had been attempted. But if a patient with nephritis be first made thoroughly warm and his skin somewhat moist by a hot-air bath, and he be kept warm by hot-water bottles, it is quite safe to apply icebags over his kidneys. Not only is it, with such precautions, quite safe, but the result is often most satisfactory.

S. S., a boy of 15, was admitted into St Mary's Hospital under my care on 9th May 1903, for sub-acute nephritis. The dropsy was slight, but the urine was reddish in colour, containing blood and epithelial and fatty casts. It was of specific gravity 1012, acid in reaction, contained albumen to the amount of one-fifth, and 1.5 per cent. of urea. The quantity passed in the twenty-four hours before treatment was begun was 34 oz. A single icebag was applied over the right kidney only, without any previous hot-air bath, as the boy's skin was warm and not dry. Directions were given that the quantity of urine passed in every successive period of eight hours should be separately measured and tested. The amount of urine on the first day of the application of the icebag was 34 oz., the same amount as on the previous day. On the second day of the ice it increased to 52 oz., on the third day it was 40 oz., on the fourth day again 52 oz. Thus the average amount of urine for the four days during which a single icebag had been applied was 44 oz., an increase of nearly 30 per cent. Two icebags were now applied, one over each kidney. A further marked increase in the quantity of urine followed immediately. On the first day of the application of two icebags the amount passed was 78 oz., on the second day 70 oz., on the third 58 oz. Thus the average amount for these three days was 68 oz., an

increase of 100 per cent. This diuresis was due solely
to the ice, for no medicine was given. The two bags
were retained in position for ten days, with the excep-
tion of one period of eight hours, the amounts passed
during these ten days being respectively 78, 70, 58,
56, 60, 53, 59, 60, 71, 70 oz., giving an average of 63.5
oz. Thus the diuresis was steadily maintained. The
quality of the urine also progressively improved. It
soon lost its red colour, though some blood-corpuscles
could still be seen when the ice was removed after
the total period of fourteen days ; the casts rapidly
diminished and entirely disappeared, and the amount
of albumen fell from about one-fifth to about one-
tenth. The amount of urea rose to 2 per cent. After
the fortnight's treatment by ice the boy was still kept
in bed for five weeks, but no other treatment was used.
The amount of albumen progressively diminished,
and finally became only a "very faint trace."

Similar results have followed in other cases, and it
is now clear that, when used with proper care, ice
over inflamed kidneys has a well-marked diuretic
influence, by diminishing the local congestion in the
inflamed organ, as it lessens the congestion in an in-
flamed appendix or in an inflamed lung. It appears
to be directly curative.

Another illustration of the diuretic effect of ice
over the kidneys in nephritis may be given.

A girl, aged 6, in my ward at the Hospital for Sick
Children, was treated for nephritis with daily hot-air
baths and aperients. During eight days of this treat-
ment the amount of urine averaged 16 oz. She slowly
improved, and when she was sent to a convalescent

home the albumen was estimated at one-third. Two weeks later she was readmitted for fresh general œdema. The amount of urine passed on the first day after her readmission was only 2 oz.; on the second day, 4 oz. A single hot-air bath was given, and then icebags were applied over the kidneys. The amount passed on the third day was $7\frac{1}{2}$ oz.; on the fourth day, 27 oz.; on the fifth, 37 oz.; on the sixth, 36 oz.; on the seventh, 37 oz.; on the eighth, 36 oz. The effect on the albuminuria was quite as remarkable. When sent to the convalescent home, the amount of albumen was estimated at one-third. When she returned, it was one-fourth. After four days of the icebag, it was only one-sixth, and two days later there was only a trace of albumen present.

One more case may be related which will show how useful the application of ice may be in a very severe case of nephritis :

H. V., aged 3 years and 10 months, was admitted into the Hospital for Sick Children on 9th November 1901, having suffered from headache, shivering, repeated vomiting for seven days, swollen face for six days, and bloody urine for two days. On admission, he was pale and very restless, with a furred tongue, and temperature of 102.8°. No urine at all was passed that afternoon and evening. He vomited once.

10th November.—Urine, 2 oz., very smoky, contains blood ; albumen $= \frac{1}{2}$. No casts. Vomited three times.

11th November.—Hot-air bath, followed by two leeches over each kidney. Hot-water bottles were placed in the bed, and two icebags were applied over the kidneys. Urine about 4 oz. (not all saved). Vomited five times. In the evening the icebags were removed for four hours. Another hot-air bath was

given. The icebags were afterwards replaced and kept in position all the night.

12th November. — Temperature, 101° to 102.2°. Urine, about 5 oz. Vomiting troublesome. Rectal feeding adopted.

13th November.—Child has been very drowsy since admission and restless. Very restless in the evening. Restlessness was increased by the hot-air bath. Temperature rose this afternoon to 104°, and a patch of dullness was found at the right base, with slight pleural friction. An icebag was placed over the right base as well as over the kidneys. Four hours later, the temperature had fallen from 104° to 102°, the pulse from 128 to 100, and the respirations from 50 to 40. The child had been sleeping a good deal. It was observed that he was much less restless while the icebags were in position, and became more restless when they were removed. Urine now contains a few hyaline and corpuscular casts, and abundant red cells.

14th November.—Total urine yesterday, 5½ oz. ; albumen, ⅓. Still vomits anything but barley-water ; retains nutrients. Temperature, 100.3° ; pulse, 100 ; respirations, 36.

15th November.—More than 10 oz. of urine yesterday. Friction at right base now not audible, but slight dullness remains. He is now taking malted milk by the mouth, and does not vomit.

16th November.—Better. Takes more interest in his surroundings. More than 13 oz. of urine yesterday.

17th November.—Last evening the temperature rose to 105.4°, and the amount of urine fell to 8 oz.

18th November.—Urine yesterday only 5 oz. Temperature, 102.8°.

19th November.—Urine yesterday more than 6 oz.

20th November. — More than 9 oz. of urine.

The temperature fell last evening to 97.6°, and the icebags were removed for eight hours; when they were reapplied it was 100.2°; at 6 P.M. it was normal. Urine contains less blood, and shows few red blood-cells, but many leucocytes with abundant epithelial and granular casts.

21st November.—Good amount of urine. Icebags left off at night, but replaced during the daytime.

25th November.—Urine in fair amount; albumen much less. Icebags discontinued altogether, after fourteen days' use.

28th November.—Urine in fair amount; cannot all be saved. It still contains leucocytes and granular and epithelial casts, but no blood, and only a trace of albumen.

5th December.—Urine contains one or two granular casts, but no leucocytes, no blood, and only a trace of albumen.

10th December.—One or two casts. Only a trace of albumen.

In conclusion, I desire to express my thanks to the Council of the Harveian Society for the honour of delivering the Harveian Lectures for 1903.

OTHER PAPERS

CASE OF MALFORMATION OF THE HEART, WITH TRANSPOSITION OF THE AORTA AND PULMONARY ARTERY.

THE heart now exhibited to the Society was taken from a male infant, 7 months old, who was under my care at the Hospital for Sick Children, Great Ormond Street, during the last four months of his life. The child had suffered from cyanosis and shortness of breath ever since its birth. When I first saw it, I found it intensely cyanosed in the head and upper limbs, decidedly less so in the trunk and lower limbs. There was, however, not the least trace of clubbing; the fingers tapered quite naturally. The heart was evidently large, and it was beating rapidly (168 in the minute); the second sound at the base was accentuated, but there was no bruit whatever. The chest in general was hyper-resonant, even down to the bases of the lungs posteriorly.

This condition continued for two months. When he was 5 months old, a bruit became developed. It was systolic, and was best heard at the left base of the heart and towards the left clavicle, also behind in the upper interscapular region. Before the child's death, however, it became much less audible, and at times seemed to have vanished.

At the autopsy it was found that two parallel vessels arose from the base of the heart. The vessel on the right side was connected with the right ventricle, and proved to be the aorta ; it gave off the coronary arteries, and, passing upwards, gave origin normally to the innominate, left carotid, and left subclavian. It then became narrowed, and joined the ductus arteriosus to form the descending aorta. The vessel on the left side, taking origin from the left ventricle, was the pulmonary artery. It was considerably larger than the aorta. It divided normally into the two branches for the lungs, and gave off a patent ductus arteriosus. Judging from the size of the pulmonary artery and from the contraction of the aorta, it would seem that the two vessels had contributed about equally to the supply of the descending aorta. The right auricle received the systemic veins, while the pulmonary veins were emptied into the left auricle. The foramen ovale was practically closed, only a very small oblique opening remaining. There was a considerable deficiency in the septum of the ventricles, and the pulmonary artery was so

placed that its entrance was above this opening; it had no doubt received blood from both ventricles. The right ventricle was hypertrophied, its walls being as thick as those of the left. The various valves were normally formed and placed, only the mitral showed some slight but distinct thickenings.

The effect on the circulation must have been that the head and upper limbs received only venous blood, while the trunk and lower limbs received blood partly arterialised. The pulmonary artery and veins must have contained almost wholly arterial blood. This peculiar circulation seems to account for the distribution of the cyanosis, and also for the dyspnœa, the respiratory centre in the medulla oblongata being furnished with none but venous blood. As to the etiology of the case, the mother stated that when about six weeks pregnant she was bathing at Hastings with three other women, when two of them suddenly sank, and were rescued with great difficulty. She was much frightened, and was ill the same day with repeated vomiting, followed by diarrhœa. Whether this be considered in any way causal of the child's condition or not, it seems, at all events, certain that a strong impression was made on the abdominal ganglia of the mother at the time of the formation of the septa of the heart, which embryologists assign as the sixth, seventh, and eighth weeks.

This case seems to throw light on the debated question of the causation of cyanosis. It was at one

time the received opinion that cyanosis was due to the intermixture of venous and arterial blood, owing to abnormal communications between the two sides of the heart.   Stillé, however, showed—1st, that cyanosis may exist without intermixture of the currents of blood; 2ndly, that there is no just proportion between the intensity of the cyanosis and the amount of venous blood which enters the systemic vessels; 3rdly, that complete intermixture may take place without cyanosis being produced; and, 4thly, that the variations in the extent, depth, and duration of the discoloration are inexplicable by the doctrine of the intermixture of the currents.   He therefore falls back upon the theory that cyanosis is due to congestion of the venous system, and points out that in fifty-three cases out of sixty-two there was obstruction or contraction of the pulmonary artery.

Dr Peacock, in his well-known work on malformations of the heart, after a careful discussion of the causation of cyanosis, comes to the same conclusion. He points out, on the one hand, that cyanosis may occur without any communication between the two sides of the heart, instancing especially the case of a cyanotic girl in whom there was an abnormal partition in the right ventricle without any other malformation; and, on the other hand, that abnormal communications are often found, and those not merely narrow, but widely open, in cases where there has been no cyanosis.   Especially remarkable are

cases such as that of Valleix, where the septum of the ventricles was so rudimentary that a complete mixture of the two blood-currents must have occurred.

These observations appear to be decisive against the theory that cyanosis is pathognomonic of the existence of abnormal openings in the heart.

Are we, therefore, obliged to fall back, as Stillé and Peacock have done, on the theory of venous congestion? The heart now exhibited seems to be an answer to this question. In this case, though there was intense cyanosis, there can have been no venous congestion. The systemic blood was returned to the right auricle and passed through a normal auriculo-ventricular opening into a well-formed and rather hypertrophied right ventricle. From this it had two means of exit, an aorta as large as an ordinary pulmonary artery, and quite unobstructed, and in addition a wide opening in the septum, which admitted it to probably quite one-third of a pulmonary artery as large as an ordinary aorta. The lungs also were fully expanded. There can, therefore, have been no venous congestion, and a confirmation of this is found in the fact that there was an entire absence of clubbing.

Hence in this case the venous congestion theory breaks down.

Is there no third theory which will account for all the cases?

I would suggest that cyanosis simply means

deficient aëration of blood, and that the amount of cyanosis is a measure of the amount to which aëration of the blood has been hindered. This is by no means the abnormal-communication theory in another form, for one may easily understand that even a considerable intermixture of venous with arterial blood would not reveal its presence if the whole mass of the blood were fairly aërated, or would only cause slight occasional lividity when other difficulties to the circulation arose, as has been the case in several recorded instances of widely patent openings and rudimentary septa. On the other hand, in such cases as the one before us the circulation is perfectly free, but aëration must have been very badly performed. The blood supplied to the head and upper limbs had not passed through the lungs at all, and some of that which passed from the right ventricle into the pulmonary artery, and by the ductus arteriosus into the descending aorta, must have been in the same condition.

Meantime, the aërated blood from the lungs was poured back into the left auricle, and thence into the left ventricle, to pass mainly into the branches of the pulmonary artery once more, being thus chased continually through the pulmonary circulation without much chance of improving by its admixture the general mass of the blood.

This same theory, a deficiency in aëration, will I think explain all cases of cyanosis. It will obviously

explain all the cases in which there has been con-
tracted or obstructed pulmonary artery or obstruction
in the right ventricle, without abnormal communica-
tions, that is to say, the cases which the venous con-
gestion theory was invented to explain. It is not the
venous congestion pure and simple, but the congestion
of non-aërated blood, that will account for them.

An objection might be brought, that on this theory
cyanosis should exist in the cases of stenosis of the
pulmonary valves in adults, but its occasional absence
here is explained by the compensatory hypertrophy
of the right ventricle.

This theory, I repeat, is not the intermixture theory
in another form ; indeed, in one point of view, it is
quite opposed to it, for, in such a case as the present,
to increase intermixture of blood would have been to
improve the aëration of the whole mass, and hence to
diminish the cyanosis. That complete intermixture
takes place in a single ventricle will matter compara-
tively little if each individual blood-corpuscle has its
turn of oxidation.

Out of Dr Peacock's twenty-five cases there are
only four in which cyanosis was not present. One of
these was a case of stenosed pulmonary valves in an
adult, which may not have been congenital. Of the
three others, one was a case of patent foramen ovale
without other defect ; the second had contraction of
the aorta with patent ductus arteriosus ; whilst in the
third both auricles opened into a single ventricle,

which supplied the pulmonary artery (the aorta springing from an atrophied right ventricle).

In all these cases there can have been no obstacle to aëration, and, accordingly, there was no cyanosis.

In one case of Dr Peacock's the presence or absence of cyanosis is not stated. All his other cases, twenty in number, were cyanotic, and in all of them there was some obstructive condition, either of the pulmonary artery or of the right ventricle ; hence in all there must have been imperfect aëration.

These cases, therefore, completely support the theory above advanced.

Clubbing of the fingers and toes is another symptom on which this case throws light. There was an entire absence of clubbing, although the cyanosis was intense. Is it not reasonable to connect this fact with the absence of congestion of the venous system ?

To test this, I have again analysed Dr Peacock's twenty-five cases. In fourteen of the twenty-five the question of clubbing is not referred to. Out of the remaining eleven, seven are said to have been decidedly clubbed, three slightly clubbed, and one probably not clubbed. In all the eleven there was obstruction on the right side of the heart ; but whereas in all the seven clubbed cases the foramen ovale was closed, in all the four cases slightly or not at all clubbed the foramen ovale was open. This seems to indicate pretty clearly that the clubbing is due to congestion of the systemic veins in cases

where the foramen ovale is closed, but that where that passage is open the congestion is so much relieved that clubbing does not result.

I will only add a reference, by way of confirmation, to Dr Pye-Smith's case of "Transposition of the Aorta and Pulmonary Artery," recorded in a previous volume of the *Transactions* (vol. xxiii. p. 80), which much resembles my case, and in which also there was cyanosis, but no clubbing ; and to one reported by Dr Crocker last year, in which, again, there was cyanosis without clubbing, and in which there was found tri-cuspid atresia, but the foramen ovale was widely open.

I submit that it is thus fairly demonstrated that cyanosis is due to defective aëration, and that clubbing is due to systemic venous congestion.

LARYNX FROM AN INFANT WHICH HAD BEEN THE SUBJECT OF A PECULIAR FORM OF OBSTRUCTED INSPIRATION.

THE larynx now exhibited to the Society was taken from an infant of a year old, which had, during its whole life, manifested a peculiar noisy respiration. Each inspiration was accompanied by a croaking sound, while expiration was much less affected (indeed, usually entirely free), and the cry-sound was quite clear. About a month before the child's death a laryngoscopic examination was made, and it was then seen that the upper aperture of the larynx was in the form of a narrow median slit, extending from above downwards, the epiglottis being folded on itself, so that the posterior surfaces of its lateral halves were almost in contact, and the ary-epiglottic folds close together and almost overlapping. A second examination was attempted a month later, when
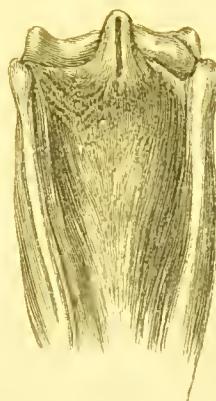
Larynx from an Infant which had been the subject of a
peculiar form of Obstructed Inspiration.

it was observed that a small white diphtheritic patch was present on each tonsil. The child died three days after this. The drawing shows the condition of the larynx as seen about two days after death.

The epiglottis is much curled inwards, even more than it usually is in infants. The aryteno-epiglottic folds are in close proximity; indeed, they seem to be in actual contact. They were quite thin, and not at all œdematous when the specimen was obtained. Above them, below the centre of the folded epiglottis, is an opening of the size of a pinhole; and below them, between the arytenoid cartilages, a second and rather larger opening. The vocal cords and the rest of the interior of the larynx were healthy. The trachea was lined with diphtheritic membrane.

I have seen four cases in which this peculiar croaking inspiration was present. All four were girls. In all the condition appeared to be congenital, and in all fairly constant, except that occasionally exacerbations seemed to be caused by exposure to cold and by flatulence. The croak is of a lower pitch than the crow of laryngismus. It continues, though less loud, during sleep, and after the administration of chloroform. There is usually some recession above the sternum during inspiration, and slightly also at the base of the thorax; but as a rule, sufficient air seems to enter the chest.

In at least one of the cases, the symptom entirely passed away as the child grew. This is, I believe, the first time that the cause of obstruction has been ascertained either by the laryngoscope or by post-mortem examination.

TWO CASES OF BRONCHOPNEUMONIA TREATED WITH BLEEDING AND ICE.

CASE I.—Emily B., a domestic servant, aged 15, but looking older, came among my out-patients at St Mary's Hospital on 24th January 1885, complaining of cough, shortness of breath, and sharp, cutting pain in the left side. She had been ill for about ten days, and thought that she had taken cold from sleeping in a damp bed, having on several occasions awaked during the night to find herself shivering. I found a small area of dullness in the second left interspace in front, and *râles* over the greater part of the left lung, and also at the base of the right. There was much dyspnœa, and the temperature was above 104°. By the kindness of Dr Cheadle, I was enabled to admit her into the hospital under my own care. She was at once put to bed, and hot poultices applied to the chest.

25th January.—She had slept fairly during the night, but this morning had much distress in breath-

ing. The dyspnœa was very obvious. There was
restlessness; the lips were livid, and the cheeks
dusky. Temperature, 105°; pulse, 136, regular, and
fairly strong; respirations, 44. The urine was of
specific gravity 1030; it contained urates, but no
albumen; chlorides were present. The bowels had
not been opened during the last four days. The
sputum was copious, viscid, not rusty. There was
dullness over the whole of the front of the left side
of the chest to the nipple-level; the breathing, how-
ever, being simply harsh, and not tubular. Resonance
was somewhat impaired below the angle of the scapula
on both sides; and at both bases, in front and behind,
was an abundance of moist *râles*, heard during expira-
tion as well as during inspiration. These physical
signs, combined with the high temperature, seemed
to denote a severe bronchitis extending to the smaller
tubes, with commencing consolidation of the left upper
lobe. Taken with the symptoms of great dyspnœa
and lividity, they seemed to render it necessary at
once to give relief to the overstrained right heart,
without waiting for the slower action of purgatives
and emetics. Venesection was, therefore, performed
at 11 A.M., 10 oz. of very dark blood being drawn
off. The relief to the dyspnœa was immediate, mani-
festing itself even while the blood was flowing; and
the lips lost their blueness. An enema was adminis-
tered, after which the bowels were opened twice. At
12.30 P.M., the temperature had fallen to 103.6°, the

pulse remaining at 136, and the respirations still in number 44, but much quieter. The patient felt much more comfortable. At 9 P.M., the temperature was again 105° ; but the lips were red, and the dyspnœa had not returned. An emetic was ordered, to clear the bronchial tubes of the very copious secretion, and a mixture, containing 10 drops of antimonial wine, and 10 gr. of bicarbonate of soda, with half an ounce of liquor ammoniæ acetatis, to be taken every four hours.

26th January.—She was relieved by the emetic, and had slept fairly well. The cough was less troublesome ; the lips were rather more blue again, and the cheeks somewhat dusky. Temperature, 105.6° ; pulse, 144 ; respirations, 48. The right side of the chest was forcibly expanded during inspiration, especially in its upper part. The left front was dull from the clavicle to the nipple-level, with bronchial breathing under the clavicle. There was dullness also about the posterior edge of the left scapula. At both bases, *râles* were heard as before. The heart was normal.

It was obvious that, though the immediate urgency had been met, the pulmonary symptoms were advancing in gravity. It was therefore determined to give up the poultices, which had been used for two days, and to try the effect of cold applications. Directions were given that the patient should be sponged, first with tepid and then with cold water. This change proving pleasant to her, an icebag was applied to

the left chest at 3 P.M.   Immediate benefit seemed
to follow.   At 8 P.M. she looked tranquil and easy ;
the lips were redder ; the temperature had fallen a
degree and a half (to 104°) ; and the frequency of the
pulse had lessened by 28 beats per minute, being now
only 116.   The respirations were still 46 per minute,
but without marked dyspnœa.   The physical signs
also had improved, there being now fair resonance
from the clavicle to the second rib.   From the second
rib to the mamma there was still dullness, with
bronchial breathing, and coarse *râle* during inspiration
only.   At the right anterior base there was still moist
râle to be heard, during both inspiration and expira-
tion.   Pain in the left side continued.

27th January.—The icebag had been kept on all
the night.   Slight delirium was observed early this
morning.   The temperature had fallen continuously,
and at 5 o'clock this morning was only 98°.   After
8 A.M., however, it rose again, and at 4 P.M. stood at
105.8°, the highest temperature throughout the illness.
Pulse, 126 ; respirations, 40.   I found that the left apex
was still improving, the resonance having now reached
as low as the third rib ; and over this area the breath-
ing was fairly normal, only the expiration a little
prolonged.   There was still dullness from the third
rib downwards, but the breathing over it was less
harsh, and there were expiratory as well as inspiratory
râles.   There were still moist sounds at the right
base as before.   So far there was improvement, but

the rise of temperature was accounted for by the dis-
covery of fine inspiratory crepitation at the angle of
the left scapula, and bronchial breathing in the axilla.
Obviously a fresh portion of lung had been attacked.
The icebag was continued, and senega substituted
for the antimonial wine.

28th January.—The temperature had again fallen
to 98°, and the pulse to 82. The respirations still
numbered 44. The dullness was now limited to a
small area at the anterior border of the left axilla,
over which loud moist *râles* were heard, with hardly
any bronchial breathing. There was diarrhœa yester-
day, the bowels being opened twelve times, and she
vomited after the medicine, which was therefore
changed to quinine. At 2 P.M., the temperature
began to rise again, but the highest point which it
attained was 103°.

29th January. — Temperature, 98° ; pulse, 108 ;
respirations, 36. She was taking food well. In the
afternoon there was another (and final) rise of tem-
perature to 104.2°. I found that the signs on the left
side were still improving, but there was now dullness
in the first interspace on the right side, which had
hitherto been quite normal, and harsh inspiration as
low as the second rib. It seemed as if the right lung
were about to follow the example of the left. Another
icebag was at once applied to the right apex. To
my surprise, I found next day (30th January) the
right apex perfectly normal, with good resonance

and natural breathing.   Pulse, 84 ; respirations, 42 ;
temperature, 96.7°.

On the 31st the temperature was 98.4°, at or about
which it remained ; the pulse, 84 ; respirations, 36.
There remained only some slight impairment of
resonance over the left lung posteriorly, and the
catarrhal sounds had quite disappeared.

From this time convalescence was uninterrupted,
and when I examined her chest, before her departure
for a convalescent home, I found everything perfectly
normal.

CASE II.—On 3rd February 1885, I was asked to
see, in consultation with Dr Langston, of Westminster,
a female infant aged 6½ months.   She had been
seriously ill for two days, and had had a slight cough
for several days previously.   She was believed to
have taken cold from exposure to cold winds.   The
temperature was 103.4°.   There was some active dis-
tension of the alæ nasi, with cough, which was
evidently painful.   On examining the chest, we found
that there was only very slight impairment of reson-
ance over the right back, with dryish *râles* over the
upper lobes behind, and a good deal of moist *râles*
over the bases, both before and behind.   We directed
that a large turpentine-stupe should be applied to the
chest, to be followed by the use of linimentum tere-
binthinæ, and that a bronchitis-kettle should be kept
constantly on the fire, the temperature of the room
being maintained at 65° F.   By the mouth, she

had been taking milligramme-granules of aconitine and of scillitine ; these were now exchanged for similar granules of emetine.

4th February.—Temperature, 103.5° (last night, 104°). There was now decided dullness over the root of the right lung, with bronchial breathing and sharp *râles*. Loud normal breathing was heard over the left lung, with some moist *râles* at the left base. She was ordered to continue the emetine, and to have large mustard and linseed poultices.

5th February, 7 P.M.—Temperature this morning, 103.5°, now 104°. The child had been very restless to-day, and cyanosed. During the afternoon, it was said to have been " quite black " around the mouth. Even by artificial light, it was easy to see that the face was dusky. The respirations were exceedingly hurried. On careful counting, there were found to be 28 inspirations in fifteen seconds, or 112 in the minute. The heart's action was comparatively slow, very little more than 100 per minute. Over the right ventricle, the second sound was loudly accentu-ated ; and even amidst the noisy inspirations, of about the same frequency as the cardiac action, the thud of the pulmonary valves could be clearly heard. It was evident that the strain on the right ventricle was rapidly becoming more than it could bear, and that, unless immediate relief were afforded, many hours would not elapse before arrest of its action would result. Three leeches were immediately sent

for.   Meanwhile, the child was placed in a tepid bath rapidly cooled.   It remained in the bath for five minutes, but the effect was to raise the rectal temperature from 104° to 105°.   Probably, a reduction of temperature would have followed a longer immersion ; but, the leeches having arrived, they were immediately applied over the sternum.   They took well, and the bleeding was afterwards encouraged by a poultice. It was estimated that the amount of blood drawn off was about an ounce, which may be considered equivalent to a moderate venesection in the adult.   It was very interesting to watch the immediate relief which followed.   Even while the leeches sucked, the breathing became much slower and deeper; indeed, the frequency of respiration sank to the rate of 50 per minute, less than one-half of its former amount.   The pulse, on the other hand, became more rapid, and was noted to be fully 120.   An hour later, the respirations numbered between 60 and 70, and the pulmonary second sound was found to be much less accentuated.   The emetine was discontinued, and it was determined to lay aside the poultices, and try the effect of external cold.   An icebag was therefore laid over the upper posterior right chest, and directions were given that milk or broth should be administered (without stimulants), and the temperature taken every hour.

6th February.—The child had slept fairly, and the cough was less troublesome.   She had taken nourish-

ment well, sucking the bottle strongly, which on the previous day she quite refused. The temperature had been over 104° all night, and for three hours was 105°. The lips and cheeks were still decidedly dusky, but nothing like so much so as the day before. The father said he "would hardly have noticed it to-day." The heart seemed now to have quite recovered itself; there was no accentuation of the pulmonary second, and the action was much more frequent, nearly 200 in the minute. The respirations were about 80. There was less dullness over the inner margin of the scapula, but perhaps a little extension of dullness outwardly, and over this spot were some sharp *râles*. During the day the temperature fell till it reached 101.7°, when the icebag was removed according to instructions. Next day (7th February) it was noted that there was distinctly less dullness over the scapula, but that at its outer edge the *râles* persisted. The left lung was now absolutely normal. In the evening, the thermometer again marked 103°, and the icebag was re-applied, but was soon removed, as the child was thought to be restless under it. The temperature, however, had fallen to 102.6° in the morning of the 8th, and to 102° in the evening.

9th February.— The temperature was now only 100.3°. The colour was much improved, no longer dusky. The cough was looser. The child lay quietly.

10th February.—A sudden accession of pyrexia

had occurred, the thermometer standing at 105.6°.
There were no new signs in the right lung, but a
patch of dullness with harsh breathing was now found
over the root of the left lung, which for the last two
days had been normal. There were also sharpish
râles at the angle of the left scapula. Respirations,
70; pulse, 170. The alæ nasi were again working
freely. There was no stress on the pulmonary
second sound. The icebag was re-applied, and an
immediate fall of temperature followed. At 8.45
A.M., when the ice was again applied, it stood at
105.4°; at 10.30 A.M., 102.6° (a fall of 3° in
less than two hours); at 12.30 P.M., 101.6°; at 2.30,
100°; at 4.30 P.M., 98.8°; at 9.30 P.M., 101°. It was
directed that the ice should be used whenever the
thermometer marked 102°.

12*th February.*— Morning temperature, 103.4°.
There was now fresh dullness over the left apex
posteriorly, with harsh breathing. Evening tempera-
ture, 101°.

13*th February.*—At 3.30 A.M., the thermometer
suddenly rose to 106°. The icebag was re-applied,
and a rapid reduction followed. At 9 A.M., it was
only 102.4°; pulse, 170; respirations, 60. The child
seemed fairly comfortable, and inclined to play with
a watch held in front of it. The dull spot at the left
apex had now quite cleared up, but there was a
finger-tip area of dullness behind the edge of the left
scapula. The *râles* had nearly disappeared.

14th February.—The physical signs in the lungs had now quite disappeared, but the temperature continued to be high (morning, 101.5°; evening, 104°). This proved to be due to the co-existence of internal otitis, resulting in posterior-basic meningitis. At all events, many of the symptoms of that disease were present. Under vigorous treatment, including para-centesis of the tympanic membranes, these symptoms entirely passed away; and, after an illness of seven weeks, the child recovered perfectly, and has since remained well.

I reserve for a subsequent communication the details of the later part of the case, which are of great interest, my object at present being simply to discuss the treatment of the pneumonia. But to prevent any misapprehension, it will be well to add that the first symptoms of the otitis were present before the icebag was first used. The aural inflamma-tion was a part of the original catarrh; and I have seen several cases in which a similar otitis of catarrhal origin (sometimes with, sometimes without, accom-panying bronchitis or pneumonia) has caused death by producing posterior-basic meningitis. But for further details on this subject, I must refer to a forthcoming paper by my colleague, Dr Barlow, and myself.

Remarks.— The true indication for bleeding in pneumonia seems to be the approach of failure of the

right heart to overcome the greatly increased pressure in the pulmonary artery, due either to extensive consolidation of lung, or to overwhelming engorgement. Evidence of this approaching failure was present in each of the cases above narrated. In the former, the necessity for bleeding was in my opinion urgent ; in the latter, it was not urgent but imperative. In both, the relief afforded was marked and immediate. It is doubtful, however, whether it would in either case have been more than temporary, but for the beneficial influence of the cold applications. The superiority of the icebag to the poultices which it replaced was very obvious in each case. The older patient was conscious of increase of comfort during its use, and the application was therefore continuous, both by day and by night. In the case of the baby, the ice was removed when the temperature sank to 102°, and replaced when a further rise occurred.

Conclusion of the second case, as reported in the *Practitioner*, August 1886 :—

The child seemed fairly comfortable, and inclined to play with a watch held in front of it. In spite of this, however, there were some fresh symptoms which caused alarm. *The head was observed to be retracted*, and on careful observation it was noticed that the respiration was distinctly tending to the Cheyne-Stokes type, exhibiting maxima and minima of frequency, with a long slow ascent and descent. The

retraction of the head was not absolutely new, for I
had observed that it was present to a very slight
degree nine days previously, and accompanied by
some stiffness of the dorsal muscles, so that it was on
that day (4th Feb.) difficult to make the child sit up
for examination of its chest.   The next day, however,
these symptoms had vanished, and no other indication
of meningeal trouble was noticed until the 13th.   The
temperature continued high, about 104° in the even-
ing and 102° in the morning.   Two days later (15th
Feb.) other symptoms of meningitis showed them-
selves.   There was occasional slight strabismus, each
eye at times diverging a little.   The pupils were
equal, but contracted (about $1\frac{1}{2}$ mm.).   Slight involun-
tary jerks of the forearms and hands were noticed.
The fontanelle was distinctly too full and tense.
Retraction of the head persisted.   Vomiting had
occurred once, but only once, and the bowels were
not constipated.   The nurse had observed sighing
several times during the last three days, also occasional
flushing of the face.   For several days also the child
had persistently raised her left hand to the side of
her head.   She now lay in a semi-comatose condition,
hardly taking any notice of surrounding objects.   The
eyelids did not close when the cornea was threatened
by the finger until it was almost touched, but they
closed when a bright light was brought.   On examina-
tion of the gums the two lower central incisors could
be felt, but they were not pressing on the gum, and

the child had not showed any sign of irritation in the mouth. It was clear to me now that the catarrh had extended up the Eustachian tube into the tympanic cavity in one or both ears, and that the resulting otitis had started, or was on the brink of starting, a basal meningitis. My experience at the Hospital for Sick Children has taught me that a form of simple basal meningitis affecting mainly the posterior part of the base of the brain, the territory supplied by the vertebral arteries, is of rather frequent occurrence in infants, and that when an opportunity for post-mortem examination occurs, muco-pus is frequently found in the tympanic cavities.

I will not enter into details about this form of meningitis, as I hope shortly to publish some observations on the subject conjointly with my friend and colleague Dr Barlow; * but I may add that the otitis is frequently a result of a previous catarrh, which has spread upwards along the Eustachian tubes.

On this occasion I explained to the parents that there was probably a collection of muco-purulent material pent up behind the tympanic membranes, which was exciting an inflammation of the brain, and advised that paracentesis of the drum-membranes should be immediately performed. They consented readily, and my colleague Mr Field, Aural Surgeon to

* *Vide* "Simple Meningitis in Children," by D. B. Lees, M.D., and T. Barlow, M.D.—*Allbutt's System of Medicine*, vol. vii., pp. 492-559.

St Mary's Hospital, performed the operation on both ears, an hour or two later.   Some blood flowed on each side, but no pus could be seen.   A poultice was then applied to each ear, and an icebag to the nape of the neck.   Two grains of iodide of potassium were ordered to be given every two hours, and mercurial ointment to be rubbed into the skin twice daily.

Almost immediately after the operation the child was observed to hold her head more erect, and two days later the retraction had entirely passed away, the fontanelle was less tense, the pupils dilated on shading, and the eyes moved conjointly.   Evening temperature, 102°.   The respiration, however, had not quite lost its Cheyne-Stokes character.   On examination of the chest, some slight loss of resonance still remained over the right scapula, and a few moist *râles* could be heard.   A leech was applied behind each ear.

For ten days after the operation she improved, and the cerebral symptoms entirely vanished.   On 25th February she had an attack of obvious earache in the left ear, with drawing up of the left shoulder.   She tore off the cotton-wool that had been placed over the ear.

A minute dose of morphia relieved her and gave her a good night's rest, but the earache recurred two days later.   The following night there was a sudden attack of dyspnœa, and the day after this I found the child again semi-comatose, with diverging eyes, and

marked and frequent spasmodic contractions of the facial muscles, and of the arms and hands.

Mr Field was again called in, and paracentesis of each drum-membrane was again performed ; fourteen days having elapsed since the first operation. After the incisions, Politzer's inflation of the tympanic cavities was practised, and a little undoubted pus was thus expelled from the left ear, blood alone flowing from the right.

The poultices to the ears were repeated, but all other treatment omitted. The ears were irrigated, and Politzer's inflation practised daily. For the second time the cerebral symptoms vanished almost immediately after the operation, and fortunately they did not return.

Small flakes of lymph-membrane appeared for several days when the left ear was irrigated, and once a piece of considerable size for the small cavity from which it came. A week after the second operation all discharge had ceased, but pain in the ears recurred, and the temperature again rose to 103.8°. Three drops of a 5 per cent. solution of cocaine were dropped into each ear, but no obvious benefit resulted. On the following day the gums were lanced over the lower incisors, which were now more prominent, and the instillation of cocaine was repeated. Next day the child looked collapsed, with a pale face, and eyes half closed. Whether the cocaine was or was not responsible for this, it is certain that she had not been

in this collapsed condition before the cocaine was used. By frequent administration of small doses of brandy and Brand's essence, she gradually revived. The earache continued. It was partially relieved by the instillation of a drop of the liquor atropinæ sulphatis into each ear, but on subsequent days this treatment proved ineffectual, and more permanent relief was obtained by washing out the ear with warm water containing a little laudanum.

Eighteen days after the second operation the left lower central incisor appeared, and it was noted that there had been hardly any earache for a day or two. The child sat up in bed and played with her toys. Three days after this, she was flushed on her right cheek and about the right ear. The gum was found to be hard over the right lower central incisor ; it was lanced, and the next day this tooth emerged. After this, convalescence was uninterrupted. The total duration of the illness was seven weeks.

Five months later (15th August) she was brought to me for inspection. She appeared in perfect health ; it was impossible to detect any evil result from the long and severe illness through which she had passed. She was now thirteen months old, could sit up naturally or crawl on the floor, was quite intelligent, and heard perfectly well on both sides. Fontanelle and circumference of head of normal size for her age.

I know that she has continued up to the present time (July 1886) in perfect health.

I

Remarks.—I think it may fairly be claimed that the life of this child was four times saved by treatment, once by the leeches, a second time by the icebag, and twice by puncture of the tympanic membranes. The leeches assuredly rescued her from imminent death; without them, she could scarcely have lived many hours longer. The recurrence of the inflammation, however, would probably have brought her again into a similar condition of peril but for the great relief given by the external application of ice. I was encouraged to adopt this treatment from observation of the immediate and striking benefit I had obtained from it in a case of bronchopneumonia in a young woman who had shortly before this been under my care at St Mary's Hospital. Within the last few days I have had another illustration of its usefulness in the case of my own youngest child, who, with a temperature of 104°, severe bronchial catarrh, and lips already growing dusky, began immediately to improve when an icebag was applied to the back of his chest, and in a few hours was quite out of danger.

With regard to the paracentesis, each operation cut short the cerebral symptoms in the most striking way. An experience of many cases enables me to state with some confidence what would have happened if meningitis had been allowed to run its course. The head-retraction would have become more pronounced, the condition of stupor permanent, and convulsions or tonic spasms might have supervened. Death would

probably have resulted ; but if an apparent recovery
had occurred, such recovery would only too probably
have been found to be incomplete, and a gradual in-
crease of the cranial circumference would have betrayed
the existence of chronic hydrocephalus. Even if life re-
mained, reason would undoubtedly have been impaired,
and the child might have proved little better than an
idiot. From such mournful alternatives of fate she was
promptly rescued by the perforation of the drum-
membranes. I have notes of two other cases in which
the same happy result has followed this operation.
In many other instances it has been ineffectual, I think
usually because it has been too late : in out-patient
practice the children are often not brought to the
hospital until the retraction of the head has lasted a
week or longer.

P.S.—January 1904. This patient is now a girl
of nineteen, healthy, intelligent, and with normal
hearing.

PRESYSTOLIC APEX-MURMUR DUE TO AORTIC REGURGITATION.

CASE I.—Henry H., aged 21 years, was admitted into St Mary's Hospital, under the care of Sir E. Sieveking, on 30th April 1887, suffering from extreme anæmia after severe and repeated epistaxis. The notes of his case were taken by Mr O. E. Higgens, M.A. He had had chorea nine years before, and rheumatic fever six years before ; he was then told that his heart was affected. After his recovery he seemed in good health, and was able to work hard ; for ten months he worked as a navvy on a railway in Canada. During the last two years he had been a French polisher, and had found no difficulty in doing his work until lately. Seven weeks before admission, while polishing a floor on his knees and with his head low, his nose began to bleed. This continued for nine hours, and it had frequently recurred for short periods. Of late, also, there had been a little shortness of breath. On admission, he was found to be very thin and extremely pallid. Pulsation was visible in all the superficial arteries. Pulse, 100 ; sudden, forcible, collapsing. The area of cardiac dullness was much increased, and the impulse diffused and visible. Four murmurs could be heard : at the base a systolic and a loud diastolic, at the apex a

presystolic and a systolic. The liver was enlarged ;
the urine albuminous, with hyaline casts and a trace
of blood. The lungs were normal. Temperature
ranged between 98.4° and 99.8°.

A few days later it was noticed that the presystolic
murmur occupied a considerable part of the diastole,
and became of a higher pitch at the end, running up
into the systole. But on 9th May it was recorded
that "the presystolic murmur observed for several
days cannot be distinctly made out to-day," though
the aortic murmurs were loud. On the same date
the spleen was felt, just below the margin of the ribs,
and some fine crackling sounds could be heard in the
left lower axillary region : the patient had complained
of a sudden sharp pain in this region the day
previously, and the temperature had risen to 101°.
The urine was pale, clear, acid, of specific gravity
1012 ; it contained albumen, granular and blood casts,
and blood-corpuscles, though not in sufficient amount
to colour it. Next day (10th May) a very large increase
of the splenic dullness was noted, and the spleen was
felt below the ribs, its lower limit being one inch
above the level of the umbilicus. The presystolic
murmur was still absent, and the systolic mitral had
developed a distinctly musical character which it had
not possessed before. But on the 11th the presystolic
was again detected, and on the 19th it was distinct,
while the systolic had vanished, the first sound at the
apex being now short and sharp. Another attack of
pain in the left side had occurred, and the spleen was
larger.

I first saw the patient on 23rd May, on succeeding
Sir E. Sieveking in charge of in-patients. There
were then a double aortic and a presystolic mitral
murmur, but the systolic mitral murmur could not
be heard. The first sound was short at the apex,
the second absent. The treatment consisted of 15-

grain doses of sulphocarbolate of sodium every four
hours, and this was continued. A few purpuric spots
developed on the patient's legs, but on the whole his
condition gradually improved; the epistaxis ceased
to recur, his strength increased, and at the end of
June he was so much better that he was allowed to
be up. The improvement continued, and on 10th
July he was permitted to go home.

On 27th July he was readmitted, on account of
recurrence of epistaxis. On 1st August I made the
following note: " Heart's impulse wavy, and diffused
over a wide area; most marked about three finger-
breadths below and two to the outer side of the nipple.
No thrill, but over the apex-beat there is a well-
marked presystolic murmur continued to the systole;
this cannot be heard to the right of the nipple-line.
At one point, just outside the impulse, a presystolic
murmur and first and second sounds can be heard.
Further to the left no murmur is audible, but a short,
forcible first sound followed by a second. The aortic
diastolic murmur is loud at the base, and is conducted
downward more toward the apex than down the
sternum; it is audible nearly to the nipple-line, but
is not audible at the xiphoid. Pulse highly character-
istic of aortic regurgitation." On the evening of this
day he had another sudden attack of pain in the
splenic region. A week later I noted that "the aortic
diastolic murmur can be traced obliquely downward
to the fifth rib, about one fingerbreadth to the
inner side of the nipple-line, and immediately below
this the presystolic murmur commences."

He was again treated with sulphocarbolate of
sodium, and with dialysed iron, but his strength
gradually failed. Recurrence of epistaxis, diarrhœa,
and increasing feebleness brought the end on 4th
September.

The post-mortem examination was made the next

day by my colleague, Dr Maguire.   The pericardium
was adherent to the heart throughout by fibrous
bands, and some similar bands passed from the front
of the pericardium to the under surface of the ribs.
The heart was much enlarged, especially the left
ventricle.   *The mitral orifice admitted three fingers*,
the tricuspid four.   All the cavities contained post-
mortem clot, and adhesive ante-mortem clot was also
found in the right auricle and in both ventricles.
The pulmonary and tricuspid valves were normal.
On the right posterior cusp of the aortic valve and
immediately below it were fresh vegetations with
ulcerations.   All the cusps were thickened from old
endocarditis.   Below the anterior cusp was a large
vegetation of fibrin adherent to a roughened inflamed
surface of endocardium apparently not ulcerated.

The mitral valve-flaps were similarly thickened,
and on the auricular surface of the anterior flap at
its middle portion, and also on the chordæ tendineæ,
were patches of recent endocarditis without ulceration.
The lungs were congested and œdematous.   There
were some old fibrous adhesions at the base of the
left pleura.   Liver large and firm.   Spleen much
enlarged, weighing fifteen ounces, its surface mottled
with small white spots.   It contained a yellow infarct
of the size of a walnut.   Both kidneys also contained
small infarcts.

As this patient died during my autumn holiday, I
was not present at the autopsy.   Nine months later,
however, I discovered that the heart had been pre-
served for the Museum, and had an opportunity of
examining it with Dr Maguire.   The weight of the
heart was now fifteen and a half ounces.   We found
that at this time the mitral orifice would not admit

more than *two* fingers, and that with some little difficulty. Dr Maguire, however, felt confident that the post-mortem record was correct, and that at the time of the autopsy the orifice admitted three fingers. Thinking that possibly the action of the spirit in which the specimen had been preserved for nine months might have caused some contraction, we examined another heart in the Museum, taken from a case of aortic aneurism with normal mitral flaps. In this case, the left ventricle being large, it is probable that the mitral orifice may have been somewhat dilated during life, yet we found that now after having been preserved in spirit, the orifice would not admit more than two fingers comfortably. It therefore seemed likely that in the other case there had been little or no stenosis, and that the post-mortem record was correct.

Case II.—James B., aged 30 years, admitted into St Mary's Hospital, under my care, 10th October 1889, suffering from dyspnœa and ascites. He was found to have a very large heart ; the impulse could be felt four fingerbreadths below and five fingerbreadths to the outer side of the nipple in the anterior axillary line.

The cardiac dullness was extensive also in the upward direction, and involved even the manubrium and the first and second intercostal spaces at the left margin of the sternum. A loud, rough, systolic murmur was heard over the whole of this basic area, and in the second right interspace close to the sternum a very local, short, diastolic murmur was detected.

In addition, the pulse-wave in the left radial artery was always smaller than that in the right. These symptoms had led to a diagnosis of aortic aneurism before his admission. Not the slightest pulsation, however, could be seen in the upper part of the thorax, and I was decidedly of opinion that the case was essentially one of aortic regurgitation. At the apex a double murmur was audible, which varied in character on different occasions. Sometimes it was systolic and diastolic, *at other times it was distinctly presystolic and systolic.* I noted, however, that the *presystolic* murmur, when it occurred, was of a blowing, not rumbling, character, and was short. At the xiphoid a tricuspid systolic murmur could be heard. The patient suffered from ascites and flatulence. The liver was enlarged, its edge reaching four finger-breadths below the costal margin ; it was firm and tender. Not much œdema of the legs. Urine of sp. gr. 1024, albuminous. The patient stated that he had never had rheumatic fever, though he had been troubled occasionally with "rheumatic pains." On the 26th of October he suddenly fell back dead.

At the autopsy it was found that the heart was very large, weighing 35 oz. with the contained clots, $27\frac{1}{2}$ oz. without them. The aortic valves were fused into a calcareous, rigid mass, occupying fully three-fourths of the orifice. An aperture of about the diameter of a cedar pencil remained ; it was situated in the left half of the normal position of the orifice, so that the stream of blood regurgitating through it must have impinged on the anterior flap of the mitral. *The mitral orifice admitted three fingers readily*, the flaps were healthy except for a very little atheroma of the base of the anterior one ; they were not shrunken or deformed. The tricuspid and pulmonary valves were normal. Liver, nutmeg and fatty. Kidneys of normal size, capsules slightly adherent.

About two inches of each radial artery was excised ;
they were equal in size, but while the vessel from the
right side was normally round, that from the left side
was distinctly flattened, as if it had long been only
partially filled. It appeared that this must have been
due to the aortic stenosis, the onward current of blood
being directed mainly toward the innominate artery.

CASE III.—William M., aged 31 years, admitted
into St Mary's Hospital 22nd October 1889. He
had never had rheumatism. Twelve years ago he
had a chancre and buboes. Three months ago he
began to have pains in his stomach, worse after meals,
and shortness of breath after exertion.

On admission there was orthopnœa, throbbing of
carotids, yellowish complexion, much dropsy of legs
and scrotum, and much albumen in the urine. Respir-
ations, 36; lungs, normal, except for some moist
sounds at the bases. The cardiac dullness was very
extensive, from the right margin of the sternum to
the left anterior axillary fold. The cardiac impulse
was diffused, being seen and felt in the fifth and
sixth interspaces, from one inch on the inner side of
the nipple-line to two inches on its outer side. At
the base a double murmur was heard, the systolic
being conducted upward, the diastolic downward
along the left side of the sternum, and loudest in the
third space. At the point of maximum impulse, in
the sixth space, two inches outside the nipple-line,
systolic and diastolic murmurs could be heard, but to
the inner side of the nipple-line, less than an inch
below the nipple, and the same distance to the right
of it, *a distinct presystolic murmur was heard. It was
decidedly rumbling in character, but it did not increase
in intensity toward its close, and did not run into a
" snap."* This presystolic murmur was not invariably
present, but it was heard three times at least during

the twelve days during which he was under observation.

Pulse, 108; somewhat collapsing in character, not full between the beats, and quite small in size.   From this smallness of the pulse and the presystolic murmur, I thought it probable that there really was mitral stenosis in this case as well as aortic incompetence, but the autopsy showed that the smallness must have been due to mitral regurgitation.   Four days before death it was reported that his urine was of specific gravity 1015, free from albumen.   He died 3rd November.

Post-mortem.—Heart very large, weight 35 oz.; all the cavities dilated.   Muscular tissue of heart normal in colour, thickness, and consistency.   The *mitral orifice admitted five fingers;* the mitral flaps normal; chordæ tendineæ normal.   Aortic valves incompetent; cusps slightly thickened, but not much deformed. Aorta highly atheromatous, in patches just above the aortic valves.   Tricuspid normal.   Pulmonary valve had only two cusps, but was otherwise normal.   The lungs contained large hæmorrhagic infarcts in the lower lobe of each and the right middle lobe.   Kidneys enlarged, each weighed 10 oz.; capsule normal, surface smooth and pale.   Liver enlarged (5 lbs. 1 oz.), section fatty and nutmeg.   Spleen, 7 oz., normal.

CASE IV.—William B., aged 45 years, admitted into St Mary's Hospital 13th May 1890.   He had never had rheumatic fever.   Stated that he had not taken much alcohol, and that he had never had syphilis.   His first symptom was swelling of the feet five weeks before admission.   Dyspnœa had been present only for one week.   On admission there was some orthopnœa.   Pulse, 100; collapsing, yet the vessel remained distended between the beats.   The cardiac dullness extended to the nipple-line, impulse feeble.

At the base a double murmur—systolic loud, diastolic not loud, and heard best in the fourth left interspace. The second pulmonary sound was accentuated. *At the spot where the apex-beat should normally be found there was a presystolic murmur, not long, but of a definitely " cantering" character.   This murmur was very local ; a little way to the left of this site it vanished,* and a systolic blowing murmur became audible.   Liver much enlarged ; no ascites.   Catarrhal sounds generally over the lungs.   The patient died six days after his admission.

Post-mortem.—Heart weighed 19 oz.   Left side empty.   Right auricle not distended.   Aortic valves quite incompetent and much diseased ; the anterior and right posterior cusps were united along their margin, so that the regurgitant stream must have been directed toward the mitral valve.   All the cusps were covered with warty vegetations, with some ulceration.   *The mitral orifice admitted four fingers ;* the tricuspid five fingers.   The anterior flap of the mitral and its chordæ tendineæ were thickened, but not shrunken ; the posterior flap was very slightly thickened.   The pulmonary and tricuspid valves were normal.   The spleen and kidney contained infarcts.

It was stated some years ago by the late Dr Austin Flint, of New York, that in certain cases of aortic regurgitation a presystolic apex-murmur might exist without any stenosis of the mitral orifice.   This statement has not met with much acceptance, even in the latest edition (1890) of the text-books on medicine.   In the interesting discussion on the " presystolic murmur, falsely so called," initiated by Dr Dickinson in the columns of the *Lancet* in

October 1887, and carried on by Dr Bristowe, Professor Gairdner, and many other distinguished physicians, it was assumed on both sides that whatever might be the true rhythm of the murmur in question, it is at all events pathognomonic of mitral stenosis. Even Dr Gairdner only inserted in a footnote the following rather sceptical reference to Dr Flint's claim: "I will observe a similar reserve as regards Dr Austin Flint's curious but exceptional experience of a murmur, apparently of mitral stenosis, going along with free aortic regurgitation, and with an uninjured mitral valve and orifice." Since that time, however, Dr Gairdner has published, in the *American Journal of the Medical Sciences* for August 1889, a case which supports Dr Flint's view, for the autopsy showed that aortic incompetence was present, and Dr Gairdner's statement implies that the mitral was normal, though he does not expressly say so.

In the *Medical Chronicle* for June 1890, my colleague, Dr Maguire, has given a summary of the cases supported by post-mortem proof of non-contracted mitral which have thus far been published. They are nine in number—three by Flint, two by Guitéras, one by Steell, one by Gairdner, and two by Osler. He adds a detailed account of two cases, one of his own and one which was under my own care, and in which he made the post-mortem examination. In the former, a typical case of aortic regurgitation, "*about one inch inside and a little above the apex-beat,*

there was heard a presystolic murmur, not rough, but rather blowing, distinctly separated from the second sound, and terminated by a normal first sound. The murmur was heard over only a limited area. . . . The presystolic murmur remained for a week, and was distinctly heard both by my colleague, Dr Cheadle, and myself. The autopsy showed that the heart was greatly enlarged, weighing 22 ounces. All its cavities were dilated. The aortic valve was markedly incompetent ; its cusps much thickened and shrunken. The thoracic aorta was extremely atheromatous and thickened. *The mitral orifice admitted easily three fingers.* The anterior flap was slightly thickened, and while its auricular surface was smooth, its ventricular surface was very slightly roughened. The posterior flap was normal, and the chordæ tendineæ were neither thickened nor shortened."

The second case was one of great pathological interest, and I therefore quote Dr Maguire's account of it in full :

H. S., aged 55 years, was admitted into St Mary's Hospital on 21st October 1887, under the care of Dr Lees, who has kindly given me permission to make use of the case. He had never had rheumatism, had lived in London all his life, and had drunk spirits and beer to excess. He had suffered for four or five·months from dyspepsia and pains in various parts, and these had increased up to the time of admission. Suppressing the immaterial details, I may relate that on examining the heart there was

seen a very diffused apex-beat, but the true apex seemed to be in the fifth interspace slightly outside the nipple-line. There were evident signs of cardiac dilatation affecting both the right and the left sides. At the aortic cartilage the first sound was weak, the second accentuated. At the fourth left interspace, near the sternum, was heard a diastolic murmur carried downward to the ensiform cartilage. *Just below the nipple a short presystolic murmur was heard*, blowing in character, and heard not quite so distinctly at the cardiac apex. The pulse was collapsing and short, and capillary pulsation was very evident. On 26th October it was noted that the presystolic murmur was much rougher in character ; that it led up to the first sound, and that it was now heard two finger-breadths outside the nipple. On 29th October no presystolic murmur was heard. In the nipple-line there was found a sharp first sound, a short systolic murmur, and a short diastolic murmur. On 31st October, in the fourth left interspace near the sternum, a short systolic and a rather long diastolic murmur were heard, but no presystolic murmur was found anywhere. At the cardiac apex there was a short systolic murmur carried for a little distance into the axilla. On 7th November the diastolic murmur was conducted down to the apex. *Thus, while the main signs of the case were those of aortic regurgitation, a distinct presystolic murmur was heard for a short time near the cardiac apex.* I made an autopsy upon this case and found the heart greatly enlarged, weighing $25\frac{1}{2}$ ozs., the enlargement being the more marked in the left ventricle. The aortic valves were incompetent and showed advanced atheroma, which had produced great shrinking of the segments. The sinuses of Valsalva above the right and anterior aortic segments were much pouched, and the artery around them was in an advanced state of atheroma. In the wall of the

posterior sinus of Valsalva an opening which admitted easily the little finger represented the orifice of the left coronary artery. The artery beyond this was transformed into a calcareous tube, somewhat larger in diameter than the opening mentioned by which it communicated with the aorta, and lay in the auriculo-ventricular groove until the anterior sulcus between the two ventricles was reached. Here the aneurism, for its nature was evident, ended by dividing into the ordinary branches of the coronary artery, which appeared to be of normal size and structure. The aneurism bulged, on its inner aspect, into the cavity of the left auricle above the anterior cusp of the mitral valve. The bulging at this spot somewhat diminished the calibre of the auricle, but it seemed to be quite clear of the cusps of the mitral valve and of the auriculo-ventricular orifice. The aneurism was empty. *The mitral orifice admitted three fingers easily.* On the anterior cusp of the mitral valve there were seen a few scattered patches of atheroma, but otherwise no abnormal appearance was noticed, and the chordæ tendineæ were healthy. All the cavities of the heart were dilated. Here, then, we had, in addition to the lesion of the aortic valve which caused its incompetence, an aneurism of the left coronary artery which caused a projection into the left auricle. Yet I think, on careful examination of the specimen, that this latter lesion could not in any way interfere with the flow of blood through the mitral orifice. Moreover, the extent of the projection could be well judged after death, for the extreme calcification of the aneurism would entirely prevent any further expansion of the vessel during life. The case seems to be truly a companion to that previously described, and thus we have two examples of presystolic murmur without mitral stenosis, and apparently the result of aortic regurgitation.

I entirely endorse Dr Maguire's description of this case, and his remarks upon it.

Adding these two cases to the four which I have above narrated, and to the nine mentioned in Dr Maguire's paper, we have fifteen cases by various observers in which a murmur of presystolic rhythm has been heard in cases of aortic regurgitation in which it has subsequently been proved by autopsy that no stenosis of the mitral existed. Cases without autopsy are of course worthless as proof, but they may be referred to as illustrations. Dr Maguire quotes one of his own and another of Dr Bramwell's, in which a presystolic murmur existed in a case of aortic regurgitation believed to be free from mitral stenosis. I have such a case under my care at present. It is a case of severe aortic regurgitation in a man of twenty-six, in which there is a typical double murmur at the base and also a rather loud systolic followed by a short diastolic at the apex. He has frequent anginal attacks. On several occasions I have heard a faint presystolic murmur to the inner side of the apex-beat, and a few days ago I noticed that it was distinctly present after the slight exertion of taking off his shirt, but that in a minute or two the rhythm changed into systolic and diastolic. On making him sit up in bed the presystolic murmur reappeared, to vanish again after a minute or two. I think it is reasonably certain that this patient has no mitral stenosis.

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I have said above, "a murmur of presystolic rhythm," for it must be confessed that it does not usually simulate very closely the rumbling, cantering sound of the murmur of mitral contraction. It is usually short and rather blowing in character. But it may partake somewhat of the well-known quality, and in Case III. this circumstance, conjoined with the smallness of the pulse, led me to diagnose a stenosis which the post-mortem examination proved to be absent. In Case I. the presystolic murmur was long and quite typical, and was even reported to have been accompanied by a thrill (this, however, was not present while the case was under my own observation). It is unfortunate that in this case the condition of the heart nine months afterwards threw some doubt on the accuracy of the record of the post-mortem examination. But if any stenosis existed, it must have been very slight, and the aortic regurgitation gave rise to the most extreme and characteristic symptoms. In the light of the other cases, I have no doubt that the presystolic murmur was in great part, if not entirely, due to the aortic regurgitation. It is worthy of note that in this case there was a large vegetation below the anterior cusp of the aortic valve, and vegetations also on and below the right posterior cusp; hence the regurgitant stream was probably mainly directed toward the left, and it would therefore strike the anterior mitral flap. Similarly, in Case IV. the anterior and right posterior cusps of the aortic

valve were actually united along their margin, leaving
the left half of the orifice alone patent ; and in Case
II. the pathological process had gone on to com-
pletely calcified union of the cusps, leaving again as
patent opening only the left portion of the normal
orifice. Hence, in all these three cases the regurgitant
stream must have impinged on the anterior mitral
flap, and there is no difficulty in seeing how this flap
was in consequence thrown into vibrations when it
was carried outward by the incoming current through
the mitral orifice at the end of the diastole. For, "in
full diastole" (to quote Dr M'Alister's account of the
observations of Ludwig and Hesse, *British Medical
Journal*, 1882, vol. ii. p. 825), "the flap and its cords
are stretched aslant across the cavity. . . . The flap
does not hang loosely down ; it is stretched taut from
basal ring to muscle-tip." Hence, under the influence
of two independent blood-currents impinging on its
opposite sides, vibrations are easily produced and give
rise to a murmur during the ventricular diastole,
more especially during its closing period, which corre-
sponds with the systole of the auricle.

THE ICEBAG AS A THERAPEUTIC AGENT.

THE value of the icebag in therapeutics is still very inadequately recognised. Tradition sanctions its employment for the arrest of hæmorrhage in hæmoptysis and in typhoid fever, though its utility in these conditions is open to question. But in visceral inflammations, with the single exception of meningitis, it has been avoided and even imagined to be dangerous. Moist warmth has been relied upon to relieve pain and to dilate the superficial blood-vessels, so that the application of poultices has long been the routine treatment of visceral inflammations, and a diagnosis of pneumonia, pericarditis, pleurisy, or peritonitis appears to the majority of practitioners an irresistible call for poultices. But the reign of the poultice has nearly ended. In surgery it has been almost banished by the antiseptic, and still more by the aseptic measures which have, during the last fifteen years, completely transformed surgical treatment. In medicine it still

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exists in some quarters as a survival—not of the fittest, for in medical cases in which moist warmth is desirable, hot moist flannels, with or without the addition of turpentine, are usually to be preferred. But it will soon, I believe, be generally recognised that many visceral inflammations ought to be treated, not with warmth, but with the local application of cold, precautions being of course taken to prevent any undue chilling of the body generally.

My first experience in the use of the icebag in pneumonia was in January 1885,* and the remarkable benefit which followed, when it replaced the poultices employed during the first two days of treatment, impressed me greatly. And in this first case I noted a fact which I have often observed subsequently, and which is of the greatest importance in an estimate of the value of this treatment—the fact that where the icebag had been applied there was produced a rapid improvement in the physical signs, although at the same time the disease was still present, and sometimes even extending, in other parts of the lung.

In the *Lancet* for 2nd November 1889, I published an account of eighteen cases of Lobar Pneumonia and Bronchopneumonia treated with the icebag, all of which recovered. In that paper I drew attention to the fact that the improvement caused by the icebag was not simply a reduction of temperature (though

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* "Two Cases of Bronchopneumonia treated with Bleeding and Ice."—*British Medical Journal*, 11th July 1885.

that often occurred to the extent of 3° or 4°), but was also a remarkable diminution of the physical signs over the diseased area and an amelioration of symptoms. I will here quote two of these cases to illustrate this statement :—

CASE XV.—Mary A——, aged 20, admitted into St Mary's Hospital 11th May 1889, on the fourth day of a pneumonia commencing at the right apex. She had a most unfavourable family history. She stated that her father suffered from asthma, that her mother had died of " galloping consumption," that she had lost ten brothers and sisters, and that, of the three who survived, two suffered from consumption. She had herself spat blood at times during the last two years. She had also suffered from " fits " for four years, and the onset of her pneumonia was marked by a fit instead of a rigor. I saw her first on the fifth day of her illness, and found evidence of pneumonia at the right apex, with temperature 104°, pulse 128, and respirations 48.

An icebag was applied over the affected apex at noon. At 6 o'clock the next morning the tempera-ture had fallen 4°, but in the course of the day it rose again to 102°. The pulse remained at 130, but the number of respirations had risen from 48 to 74, and some cyanosis had appeared. The upper part of the right lung, both in front and behind, was now dull, as far down as the angle of the scapula. On 14th May, the seventh day of her illness, the temperature was still only at 102.5°, though the pulse was nearly 130, and the number of respirations had risen to 100. The right lung seemed now to be involved in its entire extent. Both cheeks were markedly cyanosed, and the sputum, which was scanty, very viscid, and a little aërated, was of exceedingly dark colour, almost black,

the " prune-juice " expectoration admitted to be of
evil omen.   A much larger icebag was now obtained,
capable of surrounding the whole right chest, and this
was applied at 5 P.M.   Four ozs. of brandy daily were
ordered for her, and an ether and ammonia mixture
every four hours.   On the next day, the eighth, the
temperature ranged about a degree lower (101.5°), the
pulse remaining at 120, and the respirations still from
88 to 100.   But it was observed that the sputum was
distinctly less dark, and, at the same time, less viscid
and more abundant.   On the ninth day the tempera-
ture, pulse, and respiration remained about the same,
but an extraordinary improvement had occurred in
the physical signs.   There was now very fair reson-
ance over both back and front of the right lung down
to the angle of the scapula, with large moist *râles* in
front, and smaller *râles* with more natural breathing
behind.   Below the angle of the scapula there was
dullness, with fine moist *râles*, both inspiratory and
expiratory.   There was still further improvement in
the appearance of the sputum.

On the tenth morning I found that though the
temperature had been even a little higher (103°), and
was still 101°, and the pulse and respiration were
respectively 120 and 86, the improvement in the
physical signs was still more marked.

My note was, " Very fair resonance behind, even
to the base ; some impairment in the axillary region
from the posterior to the anterior axillary line.   Over
the front, resonance good as far as the nipple.   Over
the whole lung bubbling sounds can be heard, moder-
ately loud, and of double rhythm.   Cheeks bright-
coloured."   Between 6 o'clock and 10 that evening
the temperature suddenly fell from 101° to 97° ; the
crisis had arrived, and the ice-belt was removed.   After
this, convalescence was complete.

It must be allowed, I think, that in this case the

ice was of the greatest service ; it is hardly too much to say that it saved the patient's life. The condition on the seventh day, when the large ice-belt was applied, was most alarming. The entire right lung was consolidated, and the dyspnœa, the cyanosis, and the " prune-juice " expectoration indicated the gravity of the prognosis. Seventeen hours after its application a distinct improvement was observed in the sputum and in the hue of the cheeks. Next day a very extensive change for the better had occurred in the physical signs, and this improvement advanced rapidly. It had attained a most remarkable degree before the crisis occurred. It is surely uncommon in pneumonia for manifest improvement in physical signs to commence thirty-six hours before the crisis, and in this case it seemed certainly due to the local influence of the ice. The comparatively low range of the temperature throughout (after the ice was applied) should also be noticed.

CASE XVI.—Harry D——, seven years old, an inmate of the Highgate Branch of the Children's Hospital, with a retracted right chest, due to former empyema, the right lung being entirely collapsed (as was found on post-mortem examination some months later), was taken ill on 22nd June 1887. I saw him next day, and found his temperature 104°, pulse 160, respiration 56. Feeling sure, though I could not prove, that pneumonia of the left, the only working, lung was commencing, I had an icebag at once applied over it. The temperature fell 4° before the next morning, but gradually rose again, not attaining the same height, however, for forty-eight hours. On the third day of his illness I detected a small area of dullness over the root of the left lung, and at this spot bronchial breathing. He complained of pain at the epigastrium, and I noticed that his lips and cheeks

were already livid.   This was not surprising, for his other lung was useless.   The prognosis was evidently most grave, and might even have been looked upon as hopeless.   Next day, the fourth, the dullness was more extensive, being now four fingerbreadths in diameter, and albuminuria was present, but the complexion was not more blue than yesterday, and the boy seemed a little stronger.   The icebag had been persistently applied.   Pulse, 160; respiration, 60.   On the fifth day the dullness was decidedly less, measuring now only two fingerbreadths, and the temperature was lower, ranging at about 102° ; pulse, 152 ; respiration, 58.   Epigastric pain continued.   The sixth day resembled the fifth.   On the seventh day the temperature fell to normal, and the icebag was removed.

Pleuritic friction could now be heard over the dull area and below it.   The pleurisy kept his temperature a little raised for a few days, but it gradually subsided without effusion of fluid, and the boy returned to his condition before the pneumonia.   By-and-by he was able to go home, but three months later he came back to Great Ormond Street, and died there from cardiac failure.   Post-mortem examination showed that the right lung was completely collapsed, the right side of the heart greatly dilated, and the tricuspid valve incompetent, the left auricle and ventricle and mitral valve being normal.

The left lung was very voluminous ; it was healthy except for some very old cretaceous and calcified tubercle at its apex ; *there were comparatively recent pleuritic adhesions over the left lower lobe.*

In this case it is hardly possible to doubt that the icebag saved the boy's life in a condition otherwise hopeless.

Since the publication of these cases, I have continued to use the icebag in the treatment of pneumonia, and

am satisfied that in addition to its beneficial action in the reduction of temperature, it does tend to check the local inflammation of the lung.   And no difficulty need be felt in accepting this statement on the ground that pneumonia is a specific disease, due to the presence of micro-organisms, for Dr Burdon Sanderson stated in his Croonian Lectures (*British Medical Journal*, 28th November 1891, p. 1137) that "the pneumococcus is one of the most remarkable microphytes known ; first, because under certain conditions it is so extremely virulent, but secondly, because it exemplifies the general principle that virulence is one of the most variable attributes of a microphyte—*one which is most affected by its environment*."   Hence it is readily conceivable that an alteration in the environment, produced by the persistent application of cold, may be a powerful factor in checking the growth of the specific organism.   I do not, of course, claim that it will save every case of pneumonia : many of those due to influenza or alcoholism, or of septic origin, are hopeless under any kind of treatment.   But I believe that it is capable of saving some lives which would be lost if fomentations or poultices were employed, that it reduces the severity of symptoms, relieves pain, gives comfort to the patient, and brings about an earlier and a more rapid convalescence.   The relief of pain is often very striking, and not unfrequently, after the removal of the ice-bag, patients ask for its re-application, on account of the comfort they

experience from its presence. A few months ago I
saw, in consultation, a lady of 62 suffering from pneu-
monia, whose condition was critical, and growing
worse. With some reluctance, on account of her age,
I suggested that the poultices should be replaced by
an icebag, and I arranged to see her again three
hours later to watch the effect. The change was
carried out, and to my inquiry how she liked the ice,
she replied with emphasis, " It's *delicious !* " When I
removed the ice in order to examine her chest she
exclaimed, " I must have my bag again ! " Her im-
provement commenced with the application of the
ice, and she was soon convalescent.

In acute pleurisy, apart from pneumonia, the icebag
is often very helpful ; it quickly relieves pain, and
has often seemed to cut short the disease. Its action
may be aided by tightly strapping the affected side,
so as to restrain the movements of respiration, the
bag or bags containing ice being then applied over the
strapping. If the symptoms are very acute it is
useful to commence the treatment by the application
of a few leeches. When a serous effusion has occurred
into the pleural cavity, before the case came under
treatment, I have seen the use of an icebag apparently
of great service in hastening absorption.

Pericarditis I find as amenable as pleurisy to the
local influence of ice, and I have related seven cases
thus treated in a paper read at the Nottingham meet-
ing of the British Medical Association. Pain is

rapidly relieved, the extent and loudness of the friction-rub quickly diminish, and effusion is checked. The pulse becomes stronger and less frequent, the dyspnœa lessens, and it is clear that the local influence of ice on the heart in pericarditis is not depressant, but decidedly tonic. In conversation recently with Dr Leech, Professor of Therapeutics in the Victoria University, I was interested to find that he also had observed, and was much impressed by, this tonic influence of the icebag in pericarditis.

I have even seen a recent pericardial effusion rapidly absorbed beneath an icebag : in this case the diminution of the increased precordial dullness was distinctly made out within a few hours after the application of the ice, and it steadily continued. In pericarditis it is impossible to explain the improvement caused by the ice as being due to mere reduction of temperature ; for in pericarditis this is often not much raised, and it is sometimes very little depressed by the icebag which produces so much improvement in the physical signs. And if it be true that the local application of ice does diminish the violence of a pericarditis, it is a fact of the greatest possible importance in practice. Pneumonia, on recovery, leaves the lung little the worse, but pericarditis is apt to involve and damage the muscular structure of the heart, causing permanent dilatation of the cardiac cavities, especially of the right ventricle ; and a case of " cured pericarditis " is, in very many instances, a case of crippled heart.

Hence it is of the first importance to arrest a peri-
carditis as soon as possible, and from this point of
view I believe that the use of ice will be found a very
great gain.   Experience is as yet too limited to
warrant any definite statement about the after-history
of these cases ; but, from what I have already seen,
I feel confident that it will be found in the future
that the use of ice in the treatment of a case of peri-
carditis will often have the result of preventing the
loss of many years of the patient's life.

With regard to peritonitis I have little to say, but
I will point out that the local application of an ice-bag
is often of great benefit in the less acute inflamma-
tions of the vermiform appendix ("perityphlitis").   In
the more severe cases of this kind, where decided
symptoms of peritonitis are present, no time should
be lost in any palliative treatment, for such cases are
generally the result of the perforation of the appendix
by a concretion, or of a local gangrene, and if not
operated on are rapidly fatal.   I have had five cases
of this kind under my care during the last three
years : the first was not operated on, and died in
three days from the earliest symptoms ; the other
four were submitted to operation within a few hours
after admission to hospital.   All of them were found
to have the condition above described ; all four re-
covered rapidly and completely. (See *Clinical Trans-
actions*, 1892, p. 135.)

But where the inflammation of the appendix is less

acute, the local application of ice often produces very rapid relief of pain and diminution of the swelling. Any one who watches the effect of the icebag on this purely local inflammation will be prepared to accept its local influence in pericarditis and in pneumonia.

In catarrhal laryngitis the icebag quickly reduces the congestion, and thus diminishes the urgent symptoms; even in diphtheritic laryngitis it sometimes has given distinct relief.

It is not necessary for me to advocate the employment of the ice-bag in meningitis, but I should like to mention a case of posterior-basic meningitis in a young girl recently under my care at the Hospital for Sick Children, in which ice, applied to the occiput and nape of the neck, had more influence in checking obstinate vomiting than all the drugs and other means which were used.

In infantile paralysis, if seen within forty-eight hours after the onset, an icebag applied over the affected region of the spine may be expected to render good service. It is not often that these cases are brought to a hospital sufficiently early to give this treatment a chance, but I can remember one case at least in which it was apparently very successful, the resulting paralysis being very limited.

I will say nothing of the employment of ice in the treatment of orchitis, and of some cases of hernia, for of this I have no experience; but I must not omit to point out the benefit which may be obtained from it

in recent cases of sciatica.   It is now well understood
that sciatica is usually not a neuralgia but a neuritis,
that it is due to a local affection of the nerve trunk.
Hence, it is not unreasonable to expect that an icebag
applied over the inflamed part may do good.   On
two or three occasions I have seen very rapid
improvement produced in this way.   One such case I
will briefly narrate :—

Thomas A——, 27, printer, admitted into St
Mary's Hospital 2nd July 1890, for sciatica of twelve
days' duration.   On the 19th of June he had sat on a
wet seat outside an omnibus : the sciatica began next
day.   On admission, pain worst behind trochanter,
passing down thigh and leg to the foot.   Says this
limb feels numb.   He has already had four blisters,
but these have given no relief.   An icebag was applied
behind the trochanter.   The next day (3rd July)
improvement was noted ; there was less pain, and less
tenderness where the ice had been.   A second icebag
was placed over the nerve lower down.   In five days
(7th July) he was nearly well ; the tenderness behind
the trochanter and behind head of fibula had vanished ;
still "a little sore" over gluteal region above the
trochanter.   The patient was allowed to get up, and a
belladonna plaster applied.   On 12th July he was
"quite well."

Where the sciatica has lasted for several weeks one
can hardly expect much benefit from the icebag, yet
I have seen it give marked relief (not cure) in a case
of three months' standing, the patient having been
in bed for a fortnight, and having had morphine

injections three times, acupuncture three times, and eighteen flying blisters—all without benefit. The application of ice quickly "deadened" the pain, and enabled him to sleep. The improvement continued, and further benefit was obtained by massage.

In inflammatory conditions of the eye the value of iced applications is now generally recognised. My colleague, Mr Silcock, informs me that they give the greatest relief in some cases of iritis and cyclitis, and that they are frequently employed as a means of mitigating the severity of iritis after operations for cataract.

I may add a few words about the difficulties that may be encountered in endeavouring to use this method of treatment. In country districts it may be impossible to procure a supply of ice in summer; in towns it may always be obtained from a fishmonger. The block of ice needs to be broken up into small masses; this can easily be effected by means of a hammer and a pin. If an icebag is not at hand, it is usually possible to obtain a waterproof sponge-bag. Two or three new sponge-bags should be procured, and the larger the better; as a rule, two such bags are needed at once. When the bag has been loosely filled with small masses of ice, its mouth must be firmly tied, in order to prevent any escape of water. It is sometimes almost impossible to hinder this altogether, but a soft absorbent towel may be placed all round the bag. This difficulty led me to give a trial to

Leiter's coiled tubes, but I found them irksome to the patient, and not so efficient.

Another difficulty is that of keeping the icebag in its proper position, especially when the patient turns in bed. Any such movement is apt to displace the bag from its contact with the wall of the thorax, and sometimes to invert it, and thus favour the escape of water and wetting of the bed-clothes. Often it is possible to prevent these undesirable results by fixing the bag in its proper position by a few turns of a light bandage, but if there is much dyspnœa this may not be possible, and we must then rely on the carefulness and skill of the nurse, who will alter the position of the icebag when the patient moves in bed. This difficulty is less serious than might be imagined, because the soothing effect of the cold applications diminishes restlessness, and enables the patient to lie more quietly.

If there is great local tenderness which resents even the light pressure of the icebag, suspension should be tried, but in this case the nurse must take especial care to see that the suspended bag is kept actually in contact with the surface.

There is not usually any difficulty in persuading patients to allow the application of an ice-bag, and after trying it for a time they are generally well pleased with it. Twice I have known it to be thrown off after a few minutes, in the delirium of pneumonia, and occasionally the patient has objected to the

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constraint of position which it had involved, and which might probably have been avoided if he had been the sole charge of the nurse ; but as a rule the icebag gives comfort, and often it affords great relief. I can remember only one patient who, though doing well, objected to the treatment throughout.

There may sometimes be greater difficulty in private practice in persuading the friends of the patient to sanction the use of treatment so opposed to traditional notions, but, as a matter of fact, I have not in consulting practice found this to be a real difficulty. Still there is no doubt that the general practitioner must act warily in such a matter, and must remember that if recovery does not follow, he may be unjustly blamed.

Any real harm from the use of icebags may always be avoided by efficient nursing. In the case of an infant or young child the temperature should be taken hourly, and the icebag removed when the temperature falls to 100°, and replaced when it again rises to 102°. At the same time the child's legs and feet should be wrapped in hot moist flannels, and it may even be desirable to apply warm fomentations to the abdomen.

In adults also similar applications, or a hot-water bottle to the feet, are often of service, and dilatation of the cutaneous blood-vessels may be brought about by the use of such remedies as jaborandi, alcohol, and nitro-glycerine.

Special care must of course be exercised in the use of ice for aged or debilitated patients. But the case above narrated of the lady of 62, who found the icebag " delicious," shows that even at this period of life benefit may be derived from its employment. And even in such depressed conditions as influenza or alcoholism, it is possible to use this form of treatment with advantage ; and my friend Dr Sansom, of the London Hospital, lately told me of some apparently quite hopeless cases of alcoholic pneumonia under his care which had recovered after treatment with ice.

In such conditions the subcutaneous injection of strychnine will be found of considerable assistance, commencing with 2 minims of the official solution three times daily, and pushing up the dose to 6 or 8 minims, if no twitching of muscles is observed.

The length of time for which the use of the icebag should be continued in any particular case, must be decided by the progress of the disease and the general condition of the patient.

Sometimes it is desirable to use it for a few hours, and then remove it for an interval longer or shorter, as the symptoms may suggest. Thus it may be applied for four hours, then removed for a like period, and then again applied, and so on. Or it may be used for longer periods during the day, and removed at night. Each case demands a sound judgment on the part of the physician. Sometimes it may be con-

tinuously applied for a considerable period, such as two or three days or even longer, without inter-mission.

Thus, in one of the cases of pericarditis above referred to, the subject of which was a girl of 7 years of age, the icebag was kept in position over the heart during the greater part of twelve days, in fact during 186 out of the 288 hours, commencing with a continuous application of 62 hours: the child liked the icebag, and the final result was most satisfactory.

The employment of this remedy no doubt calls for care and watchfulness on the part of both nurse and physician, but with reasonable caution it involves no risk, and it is capable of rendering the most effectual service.

A CLINICAL LECTURE ON A CASE OF CHOREA AND PERICARDITIS; A CASE OF GENERAL PARALYSIS OF THE INSANE; A CASE OF MITRAL DISEASE WITH SPASMODIC DYSPNŒA; AND A CASE OF CARDIAC DILATATION.

CASE I.—*Chorea. Absent Knee-jerk. Pericarditis.*— This little girl was admitted on 17th January 1893, suffering from chorea of moderate severity. The first point worth noticing about her is that the knee-jerks were absent. This condition is occasionally found in chorea, sometimes in quite mild cases, and I am inclined to think that it is evidence of some toxic influence acting on the nerves or nerve-centres, analogous to that which undoubtedly is present in diphtheritic paralysis, and frequently in cases of diphtheria without paralytic symptoms. It suggests that the chorea itself may be due to a blood-poison acting on the cortical motor cells. The next point has reference to the heart. There was a systolic murmur at the apex, but the impulse was normal. Such a murmur in choreic children may be of a temporary character, disappearing with the chorea. No history of rheumatism could be obtained, and there were no

definite symptoms of this disease, that is to say, there was no joint affection, no nodules to be found in the usual situations, no erythema, and no tonsillitis. It was somewhat vaguely stated that her mother had suffered from "rheumatics." Such a statement, however, is but slight evidence of rheumatism, since "rheumatics" is a term loosely used by women in her position of life. No other member of the child's family had suffered from rheumatism. So far, then, there was no definite family history of rheumatism, and unless we assume that the chorea and the endocardial murmur are to be regarded as manifestations of this complaint, there is no evidence of past or present rheumatism in the child.

It was stated that she had experienced a severe fright, but further inquiry elicited the fact that this took place some months before the appearance of chorea ; it could not, therefore, have been the cause.

The choreic symptoms improved and almost vanished ; but about a month after admission she developed pericarditis ; the temperature suddenly shot up to 103°, a pericardial friction-sound could be heard, and difficulty of breathing came on, soon amounting to orthopnœa.

This case, then, is an instance of a child with chorea, in which the prominent heart-lesions of rheumatism were found without any other rheumatic signs. It is important to remember that it is by no means uncommon for children to have acute rheumatism manifested by the presence of little or nothing but the cardiac lesions due to this disease. It is also desirable to note that of the two lesions pericarditis is of infinitely more immediate importance than endo-

carditis. Later on, the valvular lesions become of moment, owing to the various other organic conditions they produce, but at their onset they are not of such grave import as pericarditis. For pericarditis causes an acute dilatation of the heart, especially of the thin-walled right cavities, by the injurious effect of the inflammation on the muscular structure. This dilatation can easily be demonstrated by the increase in the precordial dullness which it causes at a very early stage of the disease, while as yet the rub can be distinctly heard over a large part of the heart, while the impulse can be well felt, and it is clear that no great amount of fluid is present in the pericardial cavity. And this dilatation is only too apt to be permanent, partly from the effect of adhesions, but yet more from the weakening of the muscular wall, especially of the right ventricle. A case of " cured " pericarditis will often be found to have a considerable increase of precordial dullness, and a rapid pulse, usually about 120 to the minute. A child generally recovers from its first attack of pericarditis, but a second or a third attack increases the damage to the structure of the heart to such an extent as soon to cause a fatal issue. In children, it is pericarditis that kills.

If, therefore, a child presents the slightest evidence of rheumatism of any kind, whether arthritis (even the most subacute), or chorea, or nodules, or erythema, or tonsillitis, the heart must be most carefully and

frequently examined, lest pericarditis should escape observation.

To return to this case. As soon as the pericarditis was noticed, she was put upon salicylates, and an ice-bag was applied to the precordium, in accordance with the plan of treatment I have elsewhere advocated.* In two days the signs had considerably diminished ; the area of cardiac dullness was less, the friction-sound much less distinct, and the breathing easier. On the third day, as the temperature had fallen to 96°, I thought it wise to discontinue the ice-bag. A day or two later the friction-sound increased in intensity, and the temperature rose again. The icebag was re-applied for an hour or two at a time. On the fifth day the temperature rose to 102.5°, and the dyspnœa recurring, four leeches were applied. When I saw her the next morning she was in great distress ; the cardiac dullness extended two finger-breadths to the right of the sternum, and a loud rasp-ing double rub could be heard. I had the icebag re-applied, and, with a view of preventing collapse, kept the rest of the body warm by means of three hot-water bottles applied simultaneously, one to the feet, and one on each side of her. In twenty-four hours the friction-sound was much less loud, and the increase of the heart's area to the right of the sternum was considerably diminished. To-day, the fifth day since the re-application of the icebag, the area of dull-ness is about one fingerbreadth to the right of the sternum, and it extends about the same amount to the outer side of the left nipple-line, upwards to the upper margin of the third cartilage. The rub is now quite faint, and this is not owing to effusion of fluid,

* See *The Clinical Journal*, vol. i., No. 1 ; also, *British Medical Journal*, 18th February 1893.

for not only has the area of dullness diminished, but the heart's impulse can be well felt over a considerable area. There is now a short, cantering, presystolic murmur in addition to the louder systolic murmur which has been present throughout, and which is now conducted into the axilla. The pulse-rate, which was 166 before the re-application of the icebag, fell within a few hours to 132, and is to-day 106 per minute. The child is obviously better. There is no orthopnœa, and but little dyspnœa. She has nearly lost her pain, sleeps well, and plays with her toys and picture-book. The icebag is still on, and to its use I attribute the very marked improvement which has taken place.

This treatment is of special value in the early and acute stages of pericarditis. It is true that a patient thus treated needs very careful watching; but provided that the general warmth of the body is kept up, as in this case, by means of hot-water bottles, there seems to be no risk. The temperature should, however, be frequently taken, and the patient watched for signs of collapse. In the later attacks of pericarditis the application of ice is much less useful, and much more likely to produce collapse.

CASE II.—*General Paralysis of the Insane, with mainly Physical Symptoms.*—This man, 37 years of age, is interesting from a diagnostic point of view. He has been employed in the merchant service, and was lately captain of a large vessel. With the exception of syphilis when about 19, he has always enjoyed good health. He has recently had a great deal of mental anxiety as regards his pecuniary and domestic affairs. In May last, the ship he commanded was

wrecked. In the gale which caused her loss he had been exposed, and without sleep, for fifty-eight hours, as he dared not leave his post on deck. Since this misfortune he has been unable to obtain employment. He returned to England in the autumn. Some little time afterwards he began to suffer from pains in the head, and noticed that his memory was becoming impaired. Lately he has had some trouble in walking, and has double vision on looking to the right. For this last symptom he went to the Moorfields Ophthalmic Hospital, where he saw my colleague, Mr Silcock, who diagnosed his condition and kindly sent him on to me yesterday.

As there has been some difficulty in walking, we proceed first to watch his gait. You can see at once that it is unsteady, that there is distinct, though not great, ataxy. This becomes more definite if he walks with his eyes closed, and on sharply turning round he totters. When placed with his feet close together he can stand, even with closed eyes, but the movements of the tendons on the dorsum of his feet betray his insecurity. There is defect of co-ordination of the upper limbs also, for he quite fails to make the tips of his forefingers meet, even when he looks at them, and the failure is still more evident when his eyes are closed.

His pupils are unequal. They both contract a little with accommodation, but you can see that the contraction is sluggish, and the pupil soon dilates again. To light, there is little or no movement of the pupils. Movements of the eyeball show that there is weakness of both external recti muscles. The optic discs are pale, but otherwise normal.

When he is told to show his upper teeth, you note that the muscular movement is tremulous and imperfect at first, and that after one or two attempts this imperfect movement becomes impossible. Similarly,

he can whistle for a moment, but the muscular effort
of the lips soon fails.   The tongue is protruded,
straight; it is not atrophied, and is hardly at all
tremulous.

You have no doubt noticed the indistinctness of his
speech as he answered my questions : it became more
and more marked as he told his tale.   There is weak-
ness of articulatory power, increasing as he talks,
the tongue apparently being weaker than the lips, for
labials are fairly pronounced, while the tongue-sounds
are imperfect.

Now let us examine the condition of his limbs.
You see that he feels instantly, and localises correctly,
the slightest touch on the skin of his lower limbs, and
that if I move one of them without allowing him to
see what I do, he tells me at once which I have moved,
and in which direction.   He does not complain of
pains, or of any other subjective sensation.   You see
also that the nutrition of his muscles is good ; there is
nowhere any atrophy.   If you test the power of the
flexors and extensors of his knees, you find that they
are strong, and can overcome considerable resistance.
The muscles are not flabby ; on the contrary, they feel
too firm, their tone is somewhat too great, and the
effect of this is seen when we proceed to test his knee-
jerks.   On the right side we at once find a marked
increase in the jerk, but on the left side there is at
first a great difficulty in obtaining it at all.   We must
not conclude that it is absent, for you will see that the
limbs are slightly rigid, and that the patient never
thoroughly relaxes his muscles.   Even the device of
making the patient interlock his fingers and pull
forcibly fails to relax his leg-muscles while he is in a
recumbent position ; but now that we have made him
sit in a chair, and cross one leg over the other, we
succeed in a favourable moment in demonstrating
marked increase of the knee-jerk on both sides.

There is a slight tendency to ankle clonus on both sides, and the plantar reflexes are exaggerated.

What diagnosis do we form in this case? At first the ataxia and inco-ordination, and some of the eye symptoms, suggest tabes dorsalis. But the exaggeration of the knee-jerks and the absence of all sensory symptoms are against this. The rigidity and increased reflexes suggest lateral sclerosis, but the good muscular power shows that this cannot be far advanced. We have then symptoms of both these conditions, and so far as the spinal cord symptoms are concerned, we might regard the case as one of " ataxic paraplegia," that is to say, one in which both the lateral and the posterior columns are affected. But there is evidently some cerebral mischief also, as evidenced by imperfect articulation, weakness of lower facial muscles, and loss of memory. We have to deal here with a very wide range of symptoms, and in all probability we have not yet learned them all. For we have still to inquire from his friends whether he has not during the past few months shown marked deterioration of mental power, and perhaps also of moral character, which may be quite as important from the diagnostic point of view as are the physical symptoms which we see for ourselves.

Such psychical symptoms as these would throw a clear light on the meaning of his physical condition. If, in addition, delusions of grandeur were present,

the diagnosis would be quite obvious, but he appears to be entirely free from them. Such delusions, however, are not a necessary part of the disease known as general paralysis of the insane; and judging from his physical symptoms, from his impairment of memory, and his general appearance, there can be little doubt as to the nature of his disease. A cerebral tumour might cause the loss of memory, the pains in the head, and some defect of speech, but it would probably cause optic neuritis, and it could not produce this peculiar combination of spinal symptoms. These in their turn might be produced by insular sclerosis, but then there would be oscillating tremor of the upper limbs during movement, nystagmus, and a quite different affection of speech.

The causation of his disease is clear enough. The mental and physical strain of commanding a vessel in a violent gale, with no sleep and little food for fifty-eight hours, followed by the exposure and anxiety caused by the shipwreck, the disastrous effect on his own fortunes, together with pecuniary losses entailed by family difficulties, are in themselves sufficient to account for his condition. But in addition to this there is the history of syphilis. It is now quite certain that syphilis leaves behind it a predisposition to all kinds of degenerative affections of the central nervous system. The patient thinks that his syphilis was quite got rid of by iodide of potassium, but he does not know, as we do, that long after an apparent cure,

perhaps twenty or thirty years after, syphilis claims its victim. The man who has once had syphilis is never safe.

CASE III.—*Spasmodic Dyspnœa in Mitral Disease.*— This girl has mitral regurgitation, with some stenosis and probably an adherent pericardium, the results of a former attack of rheumatism, in which she had endocarditis and pericarditis. The point of special interest in her case is that she has occasional attacks of spasmodic dyspnœa and cardiac pain coming on rapidly and unexpectedly, and lasting a variable time, from half an hour to two or three hours.

Such attacks are not very rare in mitral disease, especially in mitral stenosis, and they seem to be analogous to the attacks of angina which are apt to occur when there is disease of the aortic valves. They commence without warning by dyspnœa, which rapidly increases. After ten or fifteen minutes pain is felt over the cardiac region. The dyspnœa and pain continue until the attack subsides. The patient's face becomes pale and his limbs cold; his pulse is feeble and very rapid; his respirations short and very frequent; he often sweats profusely, sometimes vomits, and occa- sionally has flatulence. These latter accompaniments seem to point to a neurosis of the vagus, while the pallor and coldness point to an imperfect filling of the left ventricle. The dyspnœa I believe to depend upon a failure of the right ventricle, accompanied in many cases if not always by an acute dilatation. In

such attacks my house-physicians have often suc-
ceeded in demonstrating a marked increase of the
cardiac dullness to the right of the sternum, and on
two or three occasions I have been fortunate enough
to be present when an attack came on, and to be able
to satisfy myself of the fact. The increase in trans-
verse dullness may amount to as much as two finger-
breadths, and I have found it return to the normal
when the attack subsided. I have also observed a
temporary tricuspid systolic murmur, which dis-
appeared when the attack was over. It is not, how-
ever, always possible to demonstrate this dilatation,
perhaps because it is often masked by distension of
the lung. A boy under my care in 1891, who had
many attacks of this kind, in several of which the acute
dilatation was clearly proved, died in a severe attack
in which the house-physician could find no increase of
the dullness. At the necropsy the lungs were found
extremely insufflated : they did not collapse on open-
ing the chest. The right side of the heart was dilated
and distended with blood. The mitral valve barely
admitted the tip of my little finger. Whenever the
right ventricle is acutely distended, dyspnœa is at once
produced. This is seen in its most acute form in em-
bolism of the pulmonary artery, and in the only case
of this affection which I have had under treatment, a
prompt venesection saved the patient's life. In less
acute, but still well-marked degree, it may be seen at
the onset of pericarditis, which rapidly produces dilata-

tion. This dyspnœa is, I believe, not merely a result of stimulation of the respiratory centre by hypervenous blood, but a true physiological reflex similar to the action exerted by stimulation of the "depressor" nerve on the vaso-motor centre. Just as the result of the latter is to open up the vaso-motor channels, and so to relieve the left ventricle, and prevent its paralysis from over-distension, so I believe a distension of the right ventricle tends to bring about its own relief by a reflex stimulus of the respiratory centre.

The symptoms pointing to vagus disturbance suggested to me the employment of atropine, which has such a marked physiological action on this nerve, and I have found it of very great service. It must, however, be given in sufficient quantity. Two minims of the official liquor atropinæ sulphatis ($= \frac{1}{50}$ grain) will usually produce little effect, but a hypodermic injection of 4 minims ($= \frac{1}{25}$ grain) will often at once cut short the attack. It has just now done so in the case of this girl. She was seized with an attack while we were investigating the case of general paralysis, and Mr Beggs promptly injected 4 minims of the solution. This was about a quarter of an hour ago, and you see that the patient is now free from distress, lying comfortably on her back, breathing fairly easily, and she states that the pain has nearly disappeared. Nitrite of amyl and nitroglycerine give some relief at times, but they are far inferior to atropine. In two or three instances where this drug has failed,

prompt relief has been given by venesection. Leeches also and dry cupping have occasionally been useful. Hypodermic strychnine and morphine are also of service.

CASE IV.—*Cardiac Dilatation. Dropsy. Action of Digitalis.*—This patient, a middle-aged woman, came into the hospital six months ago with œdema of both legs, and ascites. The area of cardiac dullness extended almost to the mid-axillary line. There was mitral and tricuspid incompetence, which passed off, so we may assume that it did not depend upon valvular disease. Under digitalis in large doses she improved very rapidly, and she was soon able to go out and follow her ordinary occupation. She returned here a month ago, with œdema of both legs, very marked ascites, and great increase of the cardiac dullness, especially to the left. The abdomen measured 48 inches in circumference, the face was cyanosed, and it was necessary to give immediate relief. One and a half pints of fluid were therefore drawn off from the abdomen by means of Southey's tubes. Ten minims of the tincture of digitalis were given every four hours, and a remarkable diuresis ensued, the amount of urine on four successive days being 74, 144, 184, and 146 ounces. The digitalis was stopped, and next day the amount of urine sank to 52 oz. and on the day following to 44 oz. As the ascites diminished, the liver was found to be enlarged, firm, and tender to the touch. It was also discovered that there was fluid in the left pleural cavity; 16 oz. were withdrawn by aspiration, giving further relief to the breathing. The area of cardiac dullness extends nearly to the anterior axillary line, but the impulse is distinct, and there is no murmur at apex or base.

What is the cause of the cardiac dilatation in this case? We know that this condition, apart from valvular lesions, may be due to pericarditis, to disease of the cardiac walls, permitting of their giving way before a normal tension, or to normal cardiac walls giving way before excessive tension, especially after sudden exertion.

There is nothing in this case which would make us attribute the dilatation to the effects of pericarditis. At first sight there appears to be no increase of arterial tension, for the pulse is easily compressible. But when we listen to the aortic second sound we find that it is distinctly a little too loud, and that to the left of the cardiac apex the second sound is undoubtedly louder than normal. These facts seem to indicate increase of arterial tension, in spite of the compressibility of the pulse. And we must remember that there are two factors concerned in arterial tension, the resistance in the arterioles and capillaries in front, and the force of the left ventricle behind. When prolonged high tension has damaged the heart to a certain degree, this organ can no longer do its share in keeping up the pressure in the arteries, and there is, consequently, a fall in tension recognisable in the pulse. Thus a low-tension pulse may mean failing heart. We come, therefore, to the question of the cause of the increase of tension in this case. The urine is of low specific gravity, contains no urates, a trace of albumen, but no casts. This strongly suggests

the presence of granular kidneys, the common cause
of increased arterial tension.   Were the albuminuria
due to the heart condition, we should expect the urine
to be of high specific gravity and to contain abun-
dance of urates.

It is probably, therefore, primarily a case of granular
kidney in which the left ventricle has yielded to the
pressure ; and it is quite likely that the ventricle-wall
itself has undergone a fibroid degeneration.

The enlargement of the liver is probably due to
passive congestion owing to back-pressure, on which
a secondary cirrhosis, an increase of fibroid tissue,
has supervened, and there may be some perihepatitis.
Of course there· may have been a primary cirrhosis,
but we have no reason to suspect the patient of
alcoholic habits.

The pleural effusion is doubtless simply a dropsy.
It is remarkable that hydrothorax, which one would
think ought to be a symmetrical condition, alike on
the two sides of the body, is frequently unilateral, as
it is in this case.   The cause of this is not evident.

From the point of view of treatment this case is
interesting.   It shows the marked relief obtained by
drawing off fluid from the abdomen, and again from
the pleura.   It illustrates also very remarkably the
beneficial action of digitalis in suitable cases.   The
very copious diuresis which resulted from it, and
which ceased .when it was discontinued, is proof of
this.   Such diuresis is brought about by an increase

in the arterial tension which digitalis effects. This increase is partly due to the action of the drug on the vaso-motor centre, partly to its effect on the muscular fibre of the heart and the arterioles. It is impossible to explain its action entirely by its influence on cardiac muscle, for physiologists find that in warm-blooded animals it does not raise arterial tension when the spinal cord is severed in the neck, and does not slow the action of the heart after section of the vagi. Hence, in warm-blooded animals it must act mainly through the nervous system, and clinically it is found often to be useless for hearts that are merely weak, and helpful to many hearts that are considerably hypertrophied if dilatation be also present. Nor is it mere rapidity of action that digitalis influences, for in neurotic tachycardia it is useless. But the rapid, irregular heart, with dilated left ventricle and low arterial tension, is always benefited by it.

IS THERE A DEXTROCARDIAC-RESPIRATORY REFLEX?

THE object of this paper is to suggest for the consideration of physiologists the question whether there is not a physiological reflex from the right ventricle of the heart to the respiratory centre, similar to that from the left ventricle along the depressor nerves to the vaso-motor centre, and with a similar function—viz., the automatic relief of over-distension of the ventricle. The suggestion is founded on the following observations of clinical phenomena.

(1) *The Dyspnœa of Pericarditis.*

I have lately had under my care at St Mary's Hospital a youth of 17 years of age, suffering from a first attack of rheumatism. He had felt rheumatic pains for about a fortnight before admission, but had been really ill for only five days. On examination, he was found to have rheumatic swelling of the wrist, and commencing pericarditis. Distinct pericardial

friction-sounds could be heard over nearly the whole
surface of the heart, especially at the base and
apex, and the absolute cardiac dullness was in-
creased, for it extended from the right border of
the sternum to half a fingerbreadth outside the left
nipple-line.   There was only the smallest possible
patch of pleurisy ; there was no pneumonia and no
valvular murmur, yet the number of respirations was
36 in the minute, the pulse being only 100, and the
temperature 102.5° F.   On inspection, the respiratory
excursions of the thorax were seen to be markedly
greater than normal.   This was not due to any paresis
of the diaphragm caused by the pericarditis, for the
epigastrium became prominent during each inspira-
tion, and the diaphragm was acting quite normally.
Nor is this all ; the sterno-mastoids were working,
and even the nostrils were dilating.   Clearly the
respiratory centre was stimulated to increased action.
What is the explanation of this phenomenon ?   There
was no obstacle to the flow of blood through the lungs
or hindrance to the access of air ; there was no
valvular disease of the heart—in short, there was
nothing but the pericarditis.   The pericardial inflam-
mation had produced dilatation of both sides of the
heart, as was evidenced by the increased percussion
dullness, and the muscular structure of each ventricle
was no doubt rendered less efficient.   The failure of
the left ventricle showed itself in the softness and
shortness of the pulse ; that of the right ventricle

produced, I would venture to suggest, the dyspnœa.
But how ?   Not, I think, by the medium of cyanosis.
The boy's face and lips were not dusky ; he had been
ill for only a few days ; and considering the absence
of any disease of the lungs, it is probable that there
was very little defect in the aëration of his blood.
How then was his very obvious dyspnœa produced ?
I would suggest that it was due to the working of
an automatic reflex from the right ventricle to the
respiratory centre, called into action by the acute
failure of the right ventricle resulting from the peri-
carditis or by irritation of the subpericardial nerves.
The same phenomenon—an early and acute dyspnœa
—occurs ordinarily in cases of pericarditis; but no
explanation of it, as far as I am aware, has been given.
It is sometimes very marked, occasionally even caus-
ing orthopnœa ; but this case presents the question
in an unusually uncomplicated manner, for there was
no affection of the lungs and almost none of the
pleura, and no previous or present valvular disease.
The dyspnœa was the result of the pericarditis and
of nothing else.

(2) *The sudden and agonising Dyspnœa of Pulmonary
Embolism.*

Three years ago a young man, 23 years of age, who
was under my care at St Mary's Hospital for pleuritic
effusion on the left side, while using the bedpan (the
nurse standing by his bedside) was suddenly seized

with great dyspnœa, became cyanosed, and called out
"Give me air, give me air!" The house-physician
was at once summoned, and found the patient *pale*, so
that he feared internal hæmorrhage had occurred.
He at once aspirated the left pleura, removing 40 ozs.
of serous fluid, and injected strychnine and digitalis
subcutaneously. Finally, the patient was wet-cupped
over the back to 4 ozs.; this seemed to give some
relief. I saw the patient four hours after the seizure;
he was then sitting up in bed, his face was ashy-livid,
and his breathing sighing (32); his pulse was com-
pressible (140). Deep inspirations could now be
heard all over both lungs, though mingled with
pleuritic rub-crackles on the left side. The cardiac
impulse was felt over a much larger area than before,
it was very forcible in the epigastrium, and was
distinctly felt to the right of the sternum. Fortun-
ately I had examined his heart on the previous day,
and had specially noted that the cardiac impulse
could not then be felt to the right of the sternum,
although the left pleura was full of fluid. Yet now,
though the fluid had been withdrawn, the impulse was
easily felt to the right of the sternum, and it was much
more violent in the epigastrium than previously. It
was clear that the right side of the heart was dis-
tended and acting strongly. There was no murmur.
Venesection to 6 ozs. was at once performed; after
this he felt easier, and there was decidedly less
violent pulsation. The next day he was much less

distressed, though the nostrils were still dilating and
the breathing was somewhat gasping ; the heart's
impulse could not be felt at all to the right of the
sternum.   On the following day he was decidedly
better ; the cardiac impulse could not be felt any-
where.   There were still rather marked expansion of
the chest, descent of the diaphragm, and dilatation of
the nostrils.   A loud, rough, pleuritic rub could be
heard all over the left side, and good entry of air
everywhere.   He left the hospital convalescent ; but
a year later he was re-admitted, dying from acute
tuberculosis.

It cannot be doubted, I think, that the cause of this
patient's agonising distress was the occurrence of a
pulmonary embolism.   The sudden check to the
aëration of the blood caused the cyanosis, which at
once became manifest, and the impeded supply of
blood to the left ventricle produced the pallor which,
combined with the cyanosis, caused the peculiar ashy-
livid tint of his complexion when I saw him.   Possibly
the cyanosis may be the explanation of the intense
dyspnœa which was at once manifested, but I was
much struck with the evidence of an acute dilatation
of the right ventricle, and with the relief that was
afforded by venesection.   The withdrawal of blood
produced marked diminution of the dyspnœa, and also
of the physical signs of cardiac dilatation, and both
improved *pari passu* during the next two days.   Now
the removal of blood could not influence the clot in

the pulmonary artery; it could only relieve the extreme pressure which the obstacle to the pulmonary circulation had caused in the right ventricle. Hence it seems to me probable that the dyspnœa was dependent, not simply on the cyanosis, but also on the overstrain on the right ventricle, and that even the intense dyspnœa of the sudden onset was due to an acute dilatation of the ventricle quite as much as to the rapid cyanosis. It seems to suggest that the great strain at once thrown on the ventricle stimulated intensely a normal reflex to the respiratory centre.

(3) *The Paroxysmal Dyspnœa of Mitral Stenosis.*

In some cases of mitral stenosis, quite apart from the chronic shortness of breath, bronchitis, pulmonary congestions, hæmorrhages, and infarcts, there occur from time to time paroxysmal attacks of dyspnœa, lasting usually for several hours if not cut short by treatment. These attacks begin with increasing difficulty of breathing, but not suddenly, and there is often a sense of oppression at the chest which may finally become distinct precordial pain. The number of respirations rapidly increases to 30, 40, 50, or even 60 per minute, the frequency of the pulse rising to 120, 150, or even 200. The patient is in great distress, and is usually very pale, often sweating profusely; not infrequently there is vomiting during the attack, occasionally flatulence, and sometimes coldness

of the limbs.   Careful percussion of the heart during such an attack will often give clear proof of the presence of temporary dilatation of the right side.   I have myself verified this on several occasions.   Two methods of treatment will often cut short such attacks : one is the withdrawal of blood by leeches or venesection ; the other is the subcutaneous injection of the thirtieth or twenty-fifth of a grain of atropine. I have several times seen an attack arrested in ten or fifteen minutes by such an injection.   As an illustration I may quote the case of a boy of 16 years of age, who was under my care at St Mary's Hospital for several months during the years 1890-91-92, whose mitral orifice at the necropsy barely admitted the tip of my little finger.   He had many attacks of dyspnœa of the kind above described, and on several occasions was rapidly relieved by atropine - injections.   On 30th December 1891, I witnessed an attack.   The boy was sitting upright in bed, his face very pale and covered with a cold sweat, taking rapid, deep inspirations, and looking like a person thoroughly exhausted from running.   The number of inspirations in the minute was 48, the pulse-rate 140.   The cardiac dullness extended from two fingerbreadths to the right of the right margin of the sternum to one fingerbreadth to the left of the left nipple-line ; his presystolic murmur had vanished, and was replaced by a short tricuspid systolic murmur.   Three minims of liquor atropinæ (B.P.) were injected.

Eight minutes later his breathing was not deeper than normal, though still 48 in the minute, and the pulse was still 140. A few hours later it was found that the limit of absolute dullness on the right had receded to the mid-sternum. The following day I saw him again, and noted that the respirations numbered only 28 and the pulse 90. I found that the absolute cardiac dullness did not pass to the right of the mid-sternum. The tricuspid murmur had vanished, and the presystolic mitral had returned. He died in a dyspnœal attack of this kind, and his lungs post-mortem were found to be extremely insufflated, not collapsing when removed, but showing no consolidation. The mitral orifice admitted only the tip of the little finger; the left ventricle was small; the right ventricle was dilated, forming the entire front of the heart, and hypertrophied, its muscular wall measuring three-tenths of an inch in thickness—precisely the same thickness as the wall of the left ventricle.

In attacks of this kind it is not always possible to demonstrate clinically the temporary dilatation of the right side of the heart, but often it is unmistakable, and the fact seems to throw a light on the causation of this paroxysmal dyspnœa. It seems to indicate an acute failure of the right ventricle to overcome the high tension in the pulmonary circuit, and to be analogous to the angina which sometimes is the result of an acute failure of the left ventricle to

overcome increased tension in the aorta.   If the
dilatation of the right heart does produce this peculiar
dyspnœa, in what way is the effect brought about?
There is no great degree of cyanosis, yet the respira-
tory centre is acting strongly, for the inspirations are
both rapid and deep.   The vomiting which not
seldom accompanies such attacks, the relief afforded
by atropine, and the fact that occasionally an attack
appears to be brought on by fright, seem to indicate
that there is a neurotic element in their causation.
But that does not consist in a spasm of the
bronchioles, for during the paroxysm there is not the
general wheezing of an attack of asthma.   The pheno-
mena appear to me to afford support to the hypothesis
that there is a nervous reflex from the right ventricle
to the respiratory centre, and that when the ventricle
fails, excitement of this reflex causes dyspnœa.

(4) *Cardiac Dyspnœa in general.*

What is the causation of ordinary cardiac dyspnœa ?
It is characterised by a sort of air-hunger, and cannot
be accounted for simply by the physical changes in
the lungs which may result from cardiac disease.   It
is often much more severe than such resulting
changes would account for, and it may exist when no
such changes can be demonstrated.   The check to
the pulmonary circulation no doubt causes a defici-
ency in the aëration of the blood, but not for a long
time sufficient cyanosis to account for the dyspnœa.

It is well recognised that in cases of mitral disease, the effectiveness of compensation depends on the vigour of action of the right ventricle. When there is marked epigastric pulsation, compensation may be effectual in mitral disease, and there may be no dyspnœa ; but if the right ventricle fails, dyspnœa is at once observed. Is this through non-aëration of blood ? Is it not more probably through excitation of a normal reflex to the respiratory centre ?

(5) *Dyspnœa of Muscular Exertion.*

Is not this due to the arrival of venous blood in the right side of the heart more rapidly than the ventricle is competent to pass it on into the lungs ? Hence the tension in the right ventricle rises, the muscular walls tend to dilate, and a stimulus probably ascends to the respiratory centre, whereby more rapid and deeper inspirations expand the pulmonary channels, and so relieve the labouring ventricle. Is not the management of the breath, which is the main part of the training for athletic exercises, simply an education of the right ventricle ?

As to the path of this hypothetical reflex, it may be suggested that its afferent part is through the superficial or the deep cardiac plexus into the vagus. Possibly an irritation of this reflex through pressure on these plexuses may be the explanation of the paroxysmal dyspnœa observed in some cases of aortic aneurism, which also, as I have seen, may

sometimes be almost immediately arrested by sub-
cutaneous injections of atropine.

It seems to me also probable that a stimulating
reflex exists from the right ventricle to the *vaso-motor
centre*, which manifests itself in the increased arterial
tension observed in mitral stenosis and in emphysema,
but which is masked in the dilatation of the right
ventricle, resulting from mitral regurgitation by the
depressing influence on the vaso-motor centre exerted
by the dilated left ventricle. Such a stimulating reflex
from the right ventricle to the vaso-motor centre would
increase the tension in the coronary arteries and thus
promote the nutrition, and therefore the strength of
contraction, of the right ventricle, in the same way as
was observed by Professors Roy and Adami after
narrowing the aorta.

ACUTE DILATATION OF THE HEART IN RHEUMATIC FEVER.

IT is not, I think, sufficiently recognised that acute dilatation of the heart is a frequent, almost a constant, occurrence in a rheumatic attack. Attention has been too exclusively directed to the auscultatory phenomena as indicative of the presence of pericarditis or endocarditis, and it seems to have escaped notice that whether either or both of these inflammatory conditions be present, or whether both be absent, there is almost always in rheumatic fever more or less dilatation of the heart. As a part of the chronic cardiac disease caused by rheumatism, dilatation is generally acknowledged to be of great importance, but it is too much regarded as merely one of the results of a valve-lesion or possibly of an adherent pericardium.

In studying the curative influence of the icebag in pericarditis, I found it necessary to determine care-

fully day by day the limits of the precordial dullness, and I was struck with the evidence of a rapid and often permanent dilatation of the heart accompanying the signs of pericarditis.   In a paper on the "Treatment of Pericarditis," published in the *Lancet*, 22nd July 1893, I attributed this early dilatation to the weakening effect of the pericardial inflammation on the cardiac muscular wall.

But observation of the fact that cardiac dilatation is much less marked in pericarditis of renal origin, or of tubercular or suppurative nature, suggested a doubt whether this explanation of the dilatation accompanying rheumatic pericarditis was altogether adequate, and the subsequent discovery of cases of rheumatism in which marked dilatation of the heart was present without any evidence of either pericarditis or endocarditis, proved that there must be some other influence at work.

In December 1894, I had under my care in St Mary's Hospital a young man of seventeen, suffering from his first attack of rheumatism, in whom the precordial dullness was greatly enlarged, without any definite murmur and without any friction or other indication of pericarditis.   I am aware that an audible rub is not invariably present in pericarditis, but the cases in which I have observed this have been of suppurative nature, and I doubt whether this complete absence of rub throughout the case ever occurs in rheumatic pericarditis.   That the increase of the

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precordial dullness was not due to effusion into the pericardium, seemed to be proved by the palpable cardiac impulse, the distinctly audible heart-sounds, and the absence of dyspnœa and distress. Under treatment, this increase in the dullness gradually became less, and returned to the normal. In 1896 I had three similar cases; in these I took tracings of the enlarged area of dullness, and again found that it diminished under treatment and gradually returned to the normal. One of these patients had been in the hospital under my care four months before (1895) with a previous attack of rheumatism, attended with great increase of the precordial dullness without either rub or murmur, and on this occasion also the excess in the dullness had disappeared on his recovery.

Before showing these tracings as evidence of alteration in the outline of the heart, one must decide whether percussion is trustworthy as a means of determining with accuracy the size of the heart. If an opinion on this matter had to be formed from the statements in textbooks, this would seem extremely doubtful; but the valuable papers of Dr Ewart in the *Lancet* of 29th August 1891, and the *British Medical Journal* of 21st March 1896, show that the true outline of the heart can be much more precisely ascertained than most physicians have supposed, while the paper read by Dr Herringham before the British Medical Association at Carlisle in July 1896, proves that the

results thus obtained are almost exactly verified by post-mortem examination.

Confusion is introduced into this question by the terms "superficial" and "deep." What is called "superficial cardiac dullness" is of no *cardiac* value whatever. It is of importance as indicating the condition of the left lung, whether on the one hand it is emphysematous, or on the other shrunken and fibrosed. But it would be much better to speak of the "uncovered cardiac area." The term "cardiac dullness" would then be understood to indicate the real size of the heart, a fact of the greatest importance.

In this way we should get rid also of the term "deep," which is apt to suggest that the percussion required to determine the true cardiac outline must be of a forcible kind. This is the very reverse of the truth, for such percussion brings out the pulmonary and gastric resonances and defeats its own object. Light percussion is absolutely necessary, especially over the sternum, which very readily conducts resonance from the lungs. If this be constantly kept in mind, there is usually little difficulty in defining with very considerable accuracy the outline of the heart on both left and right sides, for the cardiac margin on both sides is comparatively thick, and the alteration of the note on light percussion at the margin is generally quite definite.

It is well to begin in the region of the apex, and to remember that the left limit of the cardiac dullness

extends beyond, in dilatation often much beyond, the position of the impulse. The precise position of this limit should be found in the fourth and in the fifth spaces, sometimes in the sixth also.

Next, the extension towards the right of the right auricle should be determined by very careful light percussion to the right of the sternum in the fourth space, just below the line joining nipple to nipple. In the fifth space it is somewhat more difficult, because the partial hepatic dullness is here present also, but it can usually be defined satisfactorily. In health, there is always about one fingerbreadth of dullness to the right of the sternum in the fourth space, in accordance with the anatomical facts.* When the auricle is dilated, as in mitral stenosis, the limit may be two or even three fingerbreadths to the right of the sternum. This is a most valuable indication of the condition of the right auricle, and one too much neglected; it is often of great assistance in determining whether removal of blood, by venesection or by leeches, is desirable in mitral disease or in pneumonia.

I measure by fingerbreadths rather than by inches or centimetres, for the use of these measures would imply that the limit can be determined with mathematical precision, which is not the case; no more than a close approximation can be claimed. The measure by fingerbreadths is made during the

* See also the "radiographs" of the cardiac outline, by Dr Williams of Boston, *Brit. Med. Journ.*, 1898, vol. i., p. 1006.

routine examination by percussion, it is made without any marking of the patient's skin, and without exciting his attention; it is a measure which the physician always carries with him, and it is invariable for the same observer.

Having determined the extreme cardiac limit both to left and to right, the position of the lateral margins of the heart may easily be defined if it be remembered that they both slope upwards and inwards, and the percussed finger be held in a position parallel to this slope in each case. The right margin above the nipple-level rapidly approaches the sternum. The left margin normally rises to the inner side of the nipple, but in a moderately dilated heart the limit of dullness will be found to pass through the nipple, and where the dilatation is great it may cross the vertical nipple-line at one, two, in extreme cases even three, fingerbreadths above the nipple. A little practice gives facility in determining the opposite borders of the heart, so that even without any marking of the skin one seems almost to *see* the outline of the heart. But a permanent record can easily be obtained by marking the outline with a blue pencil and taking a copy of this on tracing-paper, care being taken to indicate in all cases the median line, the infra-costal angle, and the position of the nipple, as points of reference. The extension of the cardiac dullness to left and to right of the median line may then be measured in inches or centimetres.

The upper and lower limits of the cardiac outline are less easy to determine satisfactorily. Dr Ewart's observations about them, in the papers already referred to, will repay perusal. I have usually refrained from attempting to include the lower limit in the tracings, and I lay no stress on the accuracy or the precise meaning of the upper limit of the dullness, for the sternum and the great vessels here cause difficulty.

It may be well to add that the precise determination of the limit of the heart to right or to left may be impossible when there is fluid in the pleura, and it may be difficult when the breasts are large, when there is consolidation of parts of the lungs adjacent to the heart, or when emphysema exists. Fortunately, emphysema is rarely present in the subjects of acute rheumatism, who are usually in the earlier half of life. But if emphysema, pleurisy, and consolidation of lung are absent, the lateral outline of the heart obtained by careful light percussion is almost absolutely correct, as I have found by post-mortem investigation. If the outline determined by percussion be marked on the cadaver, and long needles be passed through various points in the bounding line on either side, it is found that they correspond with surprising accuracy to the margin of the heart.

It seems to me very important that a determination of its exact size by careful light percussion should invariably form a part of the routine examination of

the heart.   For it is dilatation that is the enemy, and an exact knowledge of the size and strength of the heart is far more important than the most elaborate and minute study of murmurs.   It is very necessary in typhoid fever, where the cardiac condition is of great importance ; it is indispensable in influenza and in diphtheritic paralysis, in both of which sudden death may occur from syncope ; it may save life by indicating the need for blood-letting when the right heart is becoming paralysed from over-distension in pneumonia, capillary bronchitis, or mitral disease. And surely in rheumatic fever, a disease known specially to injure the heart, it ought never to be neglected.   Yet are we not all too apt to use our stethoscope and neglect percussion, forgetting Sir George Humphry's rule, " Eyes first, fingers next, *ears last*" ?   We are prone to be satisfied if no murmur is audible.   Yet in the absence of murmur there may be great dilatation ; in proof of this I submit the following tracings.

The first three sets of tracings are taken from the cases of three young men suffering from their first or second attack of rheumatism, in none of whom any rub or definite murmur was audible, but in all of whom the cardiac dullness was for a time considerably enlarged.   In all three the cardiac dullness extended one and a half or two fingerbreadths to the left of the nipple-line, and one or one and a half fingerbreadths to the right of the sternum, on their admission into

hospital ; in all three it shrank to the normal dimensions as the rheumatic symptoms disappeared under treatment. I now show three tracings in each case, the first taken on commencing treatment, the third on recovery, and the second at an intermediate stage. Other tracings were taken, and these manifest a gradual reduction, but for the sake of clearness I show only three in each case. *Every tracing was taken without reference to previous diagrams of the same case*, so as to eliminate the element of bias as much as possible. They were carefully reduced in size on the same scale by Dr Poynton, and painted on glass, in order to make it possible to throw them on a screen by a lantern. (The first tracing in each case was coloured black, the second red, the third blue, so that when they were thrown on the screen simultaneously, the reduction in size was at once obvious.)

Of course, in all diagrams of this kind allowance must be made for some unavoidable exaggeration, due to the fact that the tracing is taken on the curved surface of the thorax and is then flattened out on a plane surface, so that the transverse diameter of the diagram is necessarily somewhat longer than the actual transverse diameter of the heart.

CASE I.—G. G—, aged 34, admitted into St Mary's Hospital 19th March 1896, suffering from slight arthritis of the left wrist and both shoulders. Temperature on admission, 100.5° ; on the next day,

99° ; on the third day, 98°.   Treatment, sodium sali-
cylate, 20 gr. every three hours.   .

G. G—, aged 34.   Rheumatic fever.
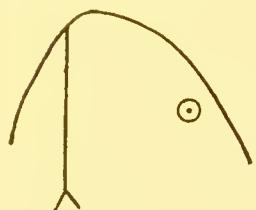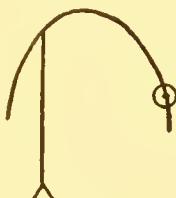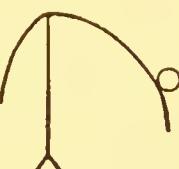
| No. 1. | No. 2. | No. 3. |
| March 19, 1896. | March 31, 1896. | April 16, 1896. |

The first tracing shows the area of cardiac dullness
on his admission.

The second tracing was taken twelve days after
the first ; the left border of the dullness has now
retreated to the nipple-line.

The third tracing was taken sixteen days after the
second, a month after the first ; the left border is now
well within the nipple-line.   The right border shows
also a return to the normal.

CASE II.—Alfred H—, aged 16 years, admitted
3rd February 1896.   Slight affection of ankles, knees,
and hip-joints.   Temperature normal.   After four
20-gr. doses of salicylate, the treatment was by 40-gr.
doses of sodium carbonate every two hours ; this was
continued for a fortnight.

The first tracing shows the cardiac area on admis-
sion.

The second tracing was taken ten days after the
first ; it shows a diminution of two fingerbreadths in

the left margin of the heart, the border now passing through the nipple.

Alfred H—, aged 16.   Rheumatic fever.

No. 1.   No. 2.   No. 3.
Feb. 3, 1896.   Feb. 13, 1896.   Feb. 20, 1896.
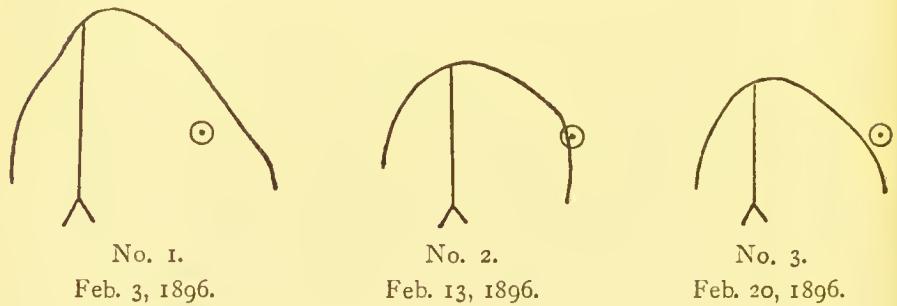
The third tracing was taken seven days after the second ; the heart has now nearly returned to its normal dimensions.

CASE III.—Albert R—, aged 21 years, admitted 6th March 1896.   Slight arthritis of left knee, ankle, and shoulder.   Temperature, 103° on first evening, 99° next morning ; some irregular pyrexia continued for fifteen days.   After about six 20-gr. doses of salicylate the treatment was by 40-gr. doses of sodium carbonate hourly during the daytime.

Albert R—, aged 21.   Rheumatic fever.

No. 1.   No. 2.   No. 3.
March 7, 1896.   March 11, 1896.   March 12, 1896.
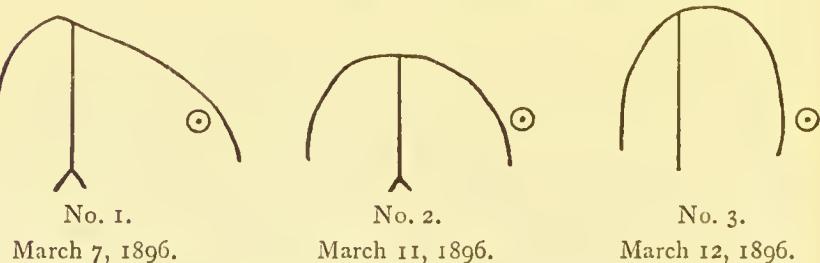
The first tracing shows the cardiac area on admission.

The second tracing was taken four days after the first, and shows a marked shifting inwards of the left edge of dullness; but the area of dullness towards the right has somewhat increased. This was apparently due to an attack of pleurisy, pleural friction being plainly heard. Leeches were applied, and in the third tracing, taken on the following day, the right border of the heart as well as the left has returned to the normal position. I have already said that I lay no stress on the upper limit of these tracings, and I think it possible that the upper limit of this third tracing may be erroneous.

This patient had been under my care four months before, during a previous attack of rheumatism. On this occasion also he had acute dilatation without pericarditis or endocarditis. At first the cardiac dullness extended to one and a half fingerbreadths, or one inch, outside the nipple-line. On his recovery it had become normal, and was found half an inch internal to the nipple.

The fourth set of tracings shows the supervention of acute dilatation in a heart already damaged by previous rheumatism.

CASE IV.—Harriet W—, aged 14 years, admitted into St Mary's Hospital 10th March 1896, suffering from her third attack of rheumatism. The first attack occurred four years before, the second three years before; in both, chorea accompanied the rheumatic symptoms. This third attack commenced three weeks ago; in it there had been dyspnœa, pain referred to the heart, also pain in the elbows and feet.

On admission, temperature, 99°; pulse, 122; respirations, 24. Fluid in both knees, ankles also swollen. Faint systolic murmur at apex, and over pulmonary artery; suspicion of presystolic murmur at apex.

Patient emotional, but not distinctly choreic. Urine faintly acid. No salicylate was employed in this case, the treatment being by 30-gr. doses of sodium carbonate given hourly during the daytime.

The first tracing was taken the day after her admission ; it shows the left border of the heart extending three fingerbreadths outside the nipple-line, and two and a half above the nipple, while the right border is fairly normal.

Harriet W—, aged 14. Rheumatic fever.
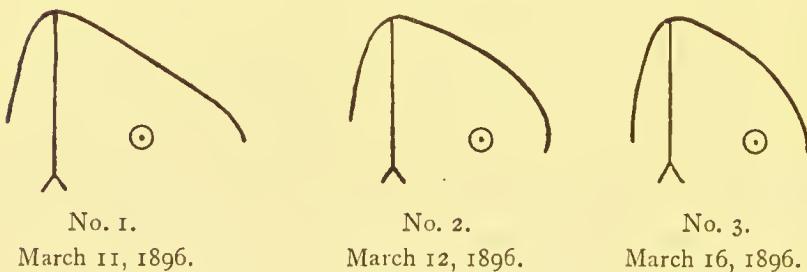
No. 1.   No. 2.   No. 3.
March 11, 1896.   March 12, 1896.   March 16, 1896.

The second tracing, taken the next day, is identical with the first on the right side, but shows a marked reduction on the left side. The presystolic murmur was now distinct.

The third tracing, taken four days after the second, is similar to the others on the right side, but shows a further large reduction on the left side. The urine was now strongly alkaline. A fourth tracing, taken nine days after the third, is practically identical with it ; she was now taking the alkaline medicine every three hours, and the urine had become again faintly acid.

The third and fourth tracings represent doubtless the permanent increase in size of her heart caused by the previous disease, which had left behind it distinct slight stenosis and regurgitation at the mitral. The

first tracing shows the additional temporary acute dilatation of the present attack of rheumatism.

The fifth set of tracings shows the effect of the combination of acute dilatation with acute pericarditis.

CASE V.—Daisy D—, aged 9 years, was admitted into St Mary's Hospital 7th February 1896, suffering from chorea, pericarditis, and slight arthritis of hands and feet. This was her second rheumatic attack. Twelve months previously she had been an in-patient for rheumatism, followed by chorea. On her admission a loud pericardial rub and a systolic apex murmur were heard by the house-physician, who at once applied an icebag over the heart, and ordered 10-gr. doses of sodium salicylate with 15 gr. of sodium carbonate every four hours. I saw her the next day and took the first diagram, which shows an enormous

Daisy D—, aged 9.   Rheumatic fever.
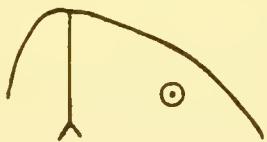
No. 1.
Feb. 8, 1896.
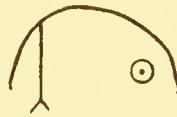
No. 2.
Feb. 21, 1896.

increase of the transverse cardiac dullness, the left border extending to more than three fingerbreadths outside the nipple-line, and about two above the nipple ; the right border also is much too far to the right. It is not unlikely that part of this increased dullness was due to the presence of some pericardial effusion. The medicine was repeated every two hours. Next day the frequency was altered to every three hours, and on the following day (10th) to every

four hours. On the 11th the urine was found to be still acid, and on the 13th it continued acid, so that the medicine was again given every three hours. On the 14th the pericardial rub had disappeared and there was no longer any chorea, but a loud systolic murmur was heard at the apex, and pleuritic friction was detected in the right axilla. The medicine was therefore given every two hours, and a second icebag was placed over the anterior base of the right lung. Two days later, the urine being still acid, the dose of sodium carbonate was doubled, and as the pleurisy had disappeared, the second icebag was removed from the right lung. On the 19th the urine had at last become alkaline. On the 22nd the ice-bag over the heart was removed, having been applied continually for fifteen days.

The second tracing was taken on the 21st, thirteen days after the first. It shows an extraordinary diminution in the cardiac dullness, and the improvement in the general condition corresponded to the improvement in the tracing. The pericarditis had subsided, but evidence of endocarditis remained. The second tracing probably indicates the permanent cardiac enlargement produced by the first rheumatic attack. The great increase in the precordial dullness shown in the first tracing may have been due in part to effusion into the pericardium, but was probably mainly due to acute dilatation.

It is worth while to point out incidentally that in this case, in spite of the pericarditis and the dilatation, the free and prolonged administration of salicylate of sodium produced no " depressing " effect.

I do not claim that these tracings are absolutely accurate ; no doubt they are open to criticism in points of detail, and I would again refer to Dr Ewart's

valuable papers. But they were taken with care, in all cases by myself, usually verified by another observer, *and always without reference to the preceding tracing*.

I think they are sufficient to prove that an acute dilatation of the heart does occur in acute rheumatism ; that it may be marked even when there is only slight pyrexia and but little arthritis, with no modification of the cardiac auscultatory phenomena beyond slight alteration in character of the heart-sounds ; and that it may complicate the evidence of former heart-disease, or of fresh endocarditis or pericarditis.

I made a preliminary statement of these facts in a letter to the *Lancet* of 25th July 1896, and also mentioned therein that a similar dilatation occurs in most cases of chorea, even when no murmur exists—a new evidence of the essentially rheumatic nature of chorea. Since that date I have made other observations on rheumatism in adults at St Mary's Hospital, and have obtained results similar to those already exhibited. I have made observations also on the occurrence of acute dilatation of the heart in the rheumatism and chorea of childhood at the Hospital for Sick Children, mainly in conjunction with my friend Dr Poynton, formerly house-physician at that hospital, and now medical registrar and pathologist at St Mary's Hospital. We have embodied our results in a joint paper, illustrated with tracings

taken very carefully by Dr Poynton and confirmed
in my own cases by myself. It seemed desirable
that the results should be compared with observations
on the hearts of children free from rheumatism and
cardiac disease. Dr Poynton therefore took tracings
of the cardiac outline in forty-five children in the
surgical wards at Great Ormond Street during his
subsequent tenure of office as house-surgeon. He
has also carefully examined the hearts of thirty-five
healthy boys, aged 12 and 13, at Marlborough School,
with the kind permission of Dr Penny. Finally, he
has analysed the post-mortem records of 150 cases of
fatal rheumatic heart-disease in children under 12.

I venture to think that the pathological and clinical
results of his researches and observations afford
additional and decisive proof of the frequency and
the serious importance of rheumatic dilatation of the
heart.

The occurrence of acute dilatation of the heart in
rheumatism has not been entirely overlooked. Thus
Dr Sansom writes (*International Clinics*, 1894, p. 7):
" I am convinced, however, that the rapid increases of
dullness over the heart in rheumatism are not all due
to pericardial inflammation and the effusion of fluid ;
the whole heart may become swollen and dilated—
swollen with the products of inflammatory exudation,
dilated because of the enfeeblement of the muscle of
its right and left chambers. In some cases this con-
dition of swollen heart disappears without any of the

friction-signs of pericarditis being manifested ; in fact, the heart and its serous membranes may pass through changes like those occurring in a joint inflamed through rheumatism. These variations in the bulk of the heart may be observed in some cases to be considerable from day to day, and there may be repeated enlargement at intervals of a few days, just as there may be repeated swelling in the joints."

Dr Samuel West reported to the Pathological Society in 1882 the case of an anæmic boy of 16, who died two months after an attack of rheumatic fever, and was found to have dilatation of all the cardiac cavities, especially of the ventricles. Evidence of endocarditis also existed. The myocardium was affected with fatty degeneration.

At a still earlier date, in 1879, Dr Goodhart exhibited to the same society the heart of a boy of 17, who died within a month after an attack which was certainly rheumatic, in whom the heart had rapidly enlarged. The left ventricle was " rather widely dilated." Weight of heart, 19 oz. ; pericardium entirely adherent by soft lymph ; recent valvulitis ; muscle slightly fatty.

In the same year, in the *Guy's Hospital Reports*, Dr Goodhart described cases of acute dilatation of the heart following scarlet fever, and another was published in 1880 by Dr Barlow in the *Medical Times and Gazette* (1880, vol. i., p. 426). In none of these, however, was there any evidence of rheumatism. But in

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a note appended to his paper, Dr Goodhart adds:
"Sir William Gull was in the habit of giving special
prominence to the fact that acute pericarditis in rheu-
matic fever is liable to give rise to rapid dilatation of
the heart, which is often mistaken for pericardial
effusion. Sir W. Gull also taught that dilatation is
succeeded, not preceded, by hypertrophy in some of
these cases." "I am very glad," Dr Goodhart con-
tinues, "to be able to mention this, not only because
it is a weighty confirmation of some of the remarks
made in this paper from another point of view, but
also because it has hitherto been unrecorded."

It may perhaps be doubted whether the value of
these observations of Sir William Gull's has been
adequately appreciated. At any rate, I think that
the frequency and extent of acute cardiac dilatation
in rheumatic fever is insufficiently recognised by most
physicians. Yet it is a condition of great gravity,
and its presence or absence ought to be carefully
ascertained in every case of the disease. When un-
complicated with pericarditis or endocarditis, if the
patient be kept absolutely at rest it may prove
transitory, and the heart may entirely recover itself,
especially under appropriate medicinal treatment.
But when it complicates pericarditis, it adds enor-
mously to the danger of the latter condition. In the
more severe cases of rheumatic pericarditis, the
accompanying acute dilatation is probably largely
responsible for the dangerous symptoms of cardiac

failure, for the dyspnœa, the tendency to cyanosis, the feeble pulse, and the delirium.

When the pericarditis is over, and has ended in pericardial adhesion, the heart becomes fixed in its dilated state, and it is never again able to return to the normal. Some amount of hypertrophy may follow, enough to maintain compensation, in the absence of much exertion, for a limited time ; but such hearts are permanently crippled, and soon break down. Cases of this kind are only too frequent in children and young adults.

Acute dilatation is probably a much more important factor than endocarditis in the production of many cases of chronic heart-disease. In a girl recently under my care at Great Ormond Street, enormous dilatation of the heart was produced in six months after the first illness. She died from pericarditis ; it was found after death that there was no fluid in the pericardial sac, but recent adhesions everywhere. There was some mitral endocarditis, but it was very slight, and entirely inadequate to account for the remarkable dilatation of the ventricles. If this patient had survived, and come under observation at a later period with an enlarged heart and a systolic apex-murmur, it is almost certain that the cardiac dilatation would have been considered the consequence of her mitral insufficiency. It is probable that very many cases of chronic heart-disease now attributed to " mitral regurgitation " or to "adherent pericardium "

are essentially the permanent results of an acute dilatation, the pericardial adhesions and the valvular damage taking but a small share in the morbid process.

It is unlikely that many cases of the acute dilatation above described are due to definite myocarditis.   No doubt this is a possible cause, and it was present in a case recently reported (with autopsy) to the Clinical Society by Dr Herringham.   But the transitory nature of the affection under appropriate treatment, as shown by the tracings now exhibited, seems to prove that in these there cannot have been any actual inflammation, though there may have been an acute congestion, of the muscle.   It is possible that skilled microscopical examination may reveal some change in the nutritive condition of the cardiac muscular fibre generally, but whether this be so or not, it seems likely that the effect is a toxic one.

Sixteen years ago (*Journal of Physiology*, vol. iii.), Dr Gaskell proved that a dilute solution of sodium hydrate caused gradual progressive contraction of the frog's ventricle until it remained persistently fully contracted and failed to relax at all ; it caused also a similar contraction of the arterioles.   On the other hand, a dilute solution of lactic acid caused relaxation and " extreme dilatation " of the ventricle, and finally diastolic standstill ; it caused relaxation also of the vessels.   He investigated the action of various drugs, and found that some of them acted like soda, others like lactic acid.

These experiments seem to suggest that the pro-
duction of the remarkable cardiac dilatation which
occurs in rheumatism may be due to the presence in
the circulating blood of a poison acting on the heart
like lactic acid. May not this be the toxin resulting
from the development of a micro-organism? It can
hardly be doubted that this is the true explanation of
the acute and sometimes fatal dilatation of the heart
which occurs in influenza. The more carefully rheu-
matism in childhood is studied, the more reason will
be found for the belief that it cannot be due to any
mere perversion of metabolism, but must be due to
some microbic process.

The suggestive observation of Dr Gaskell as to the
influence of soda in causing cardiac contraction seemed
to me to afford reason for hope that this alkali might
be found of service in diminishing the dilating effect
of the rheumatic toxin. I have therefore treated
some cases with large and frequent doses of sodium
carbonate, and I have thought that a more rapid
shrinking of the enlarged area of cardiac dullness
occurred in these cases than in those treated with
salicylates alone. The plan seems to me worthy of
further trial. At all events I found that the drug
was well tolerated in spite of its unpleasant taste, that
it never caused vomiting or lessened appetite in rheu-
matic subjects, and that large doses were required to
render the urine alkaline. I selected the carbonate
in order to secure as large an amount as possible of

the sodium element, forgetting for the time that the water of crystallisation contained in it makes it really less powerful as an antacid than the bicarbonate.   I am now making further observations upon the action of the bicarbonate.

RHEUMATIC HEART-DISEASE IN CHILDREN.

(The Introduction to a Discussion in the Section of Diseases of Children at the Edinburgh Meeting of the British Medical Association, July 1898.)

Rheumatic Carditis.

THE disease which, for want of a better term, we still call " acute rheumatism," is one of the chief destroyers of children. Those who study it only in adults can have but very inadequate ideas of its virulence in child-hood. In adults it is rare for a fatal result to be caused directly by a rheumatic attack ; in children it is much less uncommon. My friend Dr Poynton col-lected from the post-mortem records of the Hospital for Sick Children and of St Mary's Hospital 150 cases of fatal rheumatic heart-disease in children ; in nearly one-third of these (35 of 115 available) the fatal attack was the first that had occurred. He found also that of 100 cases, the clinical history of which was recorded with sufficient details, as many as 86 had exhibited symptoms of fresh rheumatism during the fatal attack,

indicating that death in the majority of instances was caused not simply by previous heart-disease, but in part by a recent toxic process.

Acute rheumatism in childhood, not so obviously and rapidly fatal as this, often does such serious damage that the patient dies during adolescence or in early adult life. This destruction is brought about by the pernicious action of the rheumatic poison upon the heart. In the Lumleian Lectures for 1894 my late colleague, Dr Sturges, drew attention to the frequency and fatality of rheumatic " carditis," and stated that of sixteen cases of this severe type which he remembered twelve died.

Endocarditis.

The fatal result is not, as a rule, due to endocarditis. Evidence of recent or of former valvulitis is indeed almost invariably found in the hearts of children dead from rheumatic heart-disease, but it is often quite slight in amount, and can have been only a minor factor. The mitral valve was implicated in all but one of the cases analysed by Dr Poynton, but in seventy-six the damage to this valve was apparently very slight, and consisted only in some thickening or small vegetations. Marked thickening and puckering of the valve segments was recorded in only three cases, and marked mitral stenosis in only nine. " Marked mitral regurgitation " was recorded in eleven cases, but it is not unlikely that in many of these it was

really due to dilatation of the orifice rather than to deformity of the valve segments.

The aortic valve is implicated much less frequently than the mitral. In the 150 cases analysed it was affected in only 51, or 34 per cent., and in only 9 of these was the affection more than slight.

Pericarditis.

Pericarditis is a much more striking phenomenon than endocarditis in the post-mortem appearances of rheumatic heart-disease in children, and probably contributes much more largely to the fatal result.   In only 9 of the 150 cases is it definitely stated that the pericardium was healthy.   It was found to be more or less adherent in 113 cases (75 per cent.), and in 77 of these, or one-half of the entire number, the adhesion was complete over the whole surface of the heart. It is clear, therefore, that more than 50 per cent. of the fatal cases have suffered from severe pericarditis, and this is doubtless a very considerable factor in the arrest of the heart's action.   How pericarditis produces this pernicious effect is well worthy of consideration.   It will probably surprise many to learn that it is rarely by the effusion of any great amount of fluid into the pericardial cavity.   The figures with reference to this are very instructive.   In only 38 cases of the 150, or 25 per cent., is it noted that any fluid at all was present in the pericardial cavity, and in many of these the amount was small.   In not more than twelve

was the amount estimated at more than 2 oz., and in only six at more than 3 oz. The highest estimates were 5 and 6 oz., and each of these in one case only.

These facts show that "pericardial effusion" in rheumatic fever is of much less frequency, amount, and importance, than would naturally be inferred from the statements of the text-books, and that the disastrous effects of pericarditis are not produced by mere effusion of fluid.

To understand how pericarditis kills, it is necessary to examine carefully the condition of the muscular wall of the heart. The visceral pericardium is really part of the heart itself ; its interstitial connective tissue is continuous with that of the cardiac muscle, and the cardiac nerves, vessels, and lymphatics course in the subpericardial tissue. It is hardly possible for the pericardium to be inflamed without some damage to the superficial part of the cardiac wall. Careful observations are greatly needed to determine whether this damage is always appreciable by the microscope, how deeply it penetrates, and whether it is more intense in the neighbourhood of the fibrous valve-ring than elsewhere. But an investigation of this kind is difficult, and a satisfactory result will probably not be attained without improvements in our present histological methods. Meanwhile, it is important to observe whether any gross changes in the cardiac muscular wall are revealed by post-mortem examination of fatal rheumatic heart-disease ; and on this matter Dr Poyn-

ton's analysis yields some valuable information. In
34 of the 150 records analysed, or 23 per cent., the
existence of some morbid condition of the myocardium
is specially mentioned. In 13 of these it was soft and
pale, in 4 fatty, in 8 tough and fibroid. These state-
ments suggest that if a careful microscopical examina-
tion had been made in each of the 150 cases, some
evidence of myocardial change would have been
detected in a considerable number.

Two important consequences may possibly follow
from the injury to the muscular wall of the heart by
the pericardial inflammation. The weakened cardiac
muscle may be stretched by the internal blood-pres-
sure, and the heart become dilated. Such dilatation,
allowing a larger quantity of blood in the ventricles
during diastole, will give the heart a harder task
during systole, and may thus lead to hypertrophy.
It becomes of interest, therefore, to ascertain whether
these results are apparent in the post-mortem records.
Dr Poynton found special mention of hypertrophy
in 58 of the 150 cases. But special mention of dilata-
tion was found in no fewer than 92 cases, and of these
56 were markedly dilated. Thus it is clear that dilata-
tion is a much more common and striking pheno-
menon than hypertrophy in the hearts of children
who have died from rheumatic heart-disease. And
it is possible that the disproportion may be even
greater than appears at first sight, for it is quite
likely that some of the " hypertrophy " may not be

genuine, but merely a thickening of the cardiac wall by œdema and inflammatory change, and thus be not compensatory, but only a further evidence of cardiac weakness.

The Main Causes of the Large Mortality.

The conclusions to be drawn from this analysis seem to be that endocarditis and pericardial effusion have very little share in the production of the fatal result in the rheumatic heart-disease of early life, and that the main factors responsible for the mortality and for a large part of the cardiac crippling in those who survive are (1) pericarditis of plastic type; and (2) dilatation of the heart.

These facts ought to be carefully kept in mind when we proceed to examine clinically the heart of a child suffering from rheumatism. Let us remember that murmurs are of comparatively small importance in the immediate prognosis, and that the points which it is essential to determine are: (1) evidence of pericardial friction; and (2) evidence of cardiac dilatation. With regard to the first of these points, it is hardly necessary, before the present audience, to say anything.

Cardiac Dilatation.

But it seems desirable to direct special attention to the question of cardiac dilatation. I have hitherto spoken of this only in connection with pericarditis,

and it certainly often accompanies that condition. I discovered this for myself while studying the curative influence of the icebag in pericarditis, not then knowing that it had been pointed out by Sir William Gull years ago. It seemed at first easy to explain the dilatation by the weakening effect of pericarditis on the cardiac muscle. But further study soon showed that this was not the complete explanation, for I found that more or less dilatation is usually present in a rheumatic attack in which there is no proof of either pericarditis or endocarditis ; in subacute first attacks with slight pyrexia and little arthritis ; in adults as well as in children. By careful light percussion it is generally easy to demonstrate a decided increase in the area of deep cardiac dullness, especially towards the left, and a shifting of the left margin of the heart outwards, so that it passes no longer within the nipple-line, but through the nipple, or outside and above it. The cardiac impulse is enfeebled and diffused, and the site of the maximum impulse shifted towards the left, into the nipple-line, or even external to it, though not so far to the left as the left margin of dullness. The first sound becomes somewhat altered in character. There may be no murmur at all, or only a faint blowing systolic murmur over the right ventricle, such as occurs not rarely in apparently healthy children.

Tracings shown by Dr Poynton and myself at the last meeting of the Royal Medical and Chirurgical

Society, and to be published in the next volume of its *Transactions*, show that in all there is evidence of dilatation of the left side of the heart, sometimes of the right side also ; that the dilatation tends to diminish as the rheumatic symptoms subside, and that it may increase again if the rheumatism relapses.

It is clear from evidence of this kind that dilatation of the heart is a common phenomenon in rheumatism, and that it is by no means limited to the cases which suffer from pericarditis, though it may become much more pronounced when pericarditis is present. It appears to be due to a toxic action of the rheumatic poison on the cardiac muscle. A similar acute dilatation occurs in influenza, and is often the cause of severe symptoms, sometimes of a fatal result. Both in influenza and in rheumatism the cardiac dilatation caused by the acute attack may remain when the attack is over, gradually producing symptoms of increasing cardiac failure, and death a few years afterwards. It cannot be doubted that in influenza the deleterious action on the heart is the effect of a toxin produced by a microbic growth ; it seems highly probable that the same is true for rheumatism.

A child who has once suffered from rheumatism is very liable to have subsequent attacks. Often the dilatation caused by the first attack has not subsided when the second attack occurs, and causes further dilatation. The heart may thus become of very large size ; the dullness may extend as far as three or even

four fingerbreadths to the left of the nipple, the left
margin crossing the nipple-line two or even three
fingerbreadths above the nipple ; and it may extend
two or even three fingerbreadths to the right of the
sternum in the fourth right space. Upwards it may
extend as high as the upper border of the second left
costal cartilage, without any pericardial effusion.

Pericardial Effusion.

Sometimes, doubtless, the presence of more or less
fluid in the pericardial cavity further increases the
precordial dullness, but usually the major part of the
enlargement, and often the whole of it, is due to
dilatation. The figures quoted above show how
rarely any considerable quantity of fluid is found in
the pericardial cavity after death. Is it possible to
determine clinically with any certainty whether peri-
cardial effusion is present in addition to the dilatation ?
In this connection Dr Sansom lays stress on a con-
siderable extension of dullness in the third and second
intercostal spaces. This sign is of value, for the fibro-
serous inflammatory product of early rheumatic peri-
carditis is most abundant at the base of the heart,
around the great vessels. It has been suggested that
dullness in the fifth right space is indicative of fluid
in the pericardium, but this is certainly incorrect, for
it may be caused by dilatation of the right auricle.
Dr Ewart thinks that fluid in the pericardial cavity
distends the pericardial sac at its attachment to the

diaphragm, and that the right and left margins of the precordial dullness then slope outwards as they descend, meeting the horizontal line of absolute liver dullness at an acute angle. The fact of cardiac dilatation accompanying rheumatic pericarditis makes this point very difficult to determine with accuracy, and practically deprives it of value. Nor has any post-mortem evidence been adduced to prove that the presence of fluid in the pericardium could produce such angular outlines.

In the treatment of an acute rheumatic attack it is extremely important to remember that a rapid increase in the area of precordial dullness, which is very liable to be attributed to effusion into the pericardium, is mainly due to acute dilatation of the heart, and may be wholly due to it.

Chronic Dilatation.

In chronic heart-disease resulting from former rheumatic attacks, chronic cardiac dilatation, the permanent result of a former acute dilatation, plays a large part. If during life there is evidence of adhesion of the pericardium, or if such adhesion is found after death, the damage to the heart is usually attributed to "adherent pericardium." When external pericardial adhesions exist, fixing the heart to the sternum, pleuræ, and lungs, there can be little doubt that these have an injurious effect, especially if they at all constrict the great vessels. But it is

doubtful whether simple adhesion of the two peri-
cardial surfaces does much harm, except in this
respect : that it tends to render permanent an acute
dilatation and to hinder its diminution.

Systolic and Presystolic Murmurs.

If, again, as is often the case, along with the per-
manent dilatation, a systolic murmur at the apex is
audible, the symptoms are usually thought to be due
to " mitral regurgitation " pure and simple.   Now it
can hardly be doubted that a persistent and unvary-
ing systolic murmur at the apex, conducted towards
the axilla, is good evidence of more or less regurgita-
tion through the mitral orifice, and it seems certain
that mitral regurgitation, by causing increased intra-
auricular tension, and therefore increased intraven-
tricular tension during diastole, must tend to produce
some dilatation and hypertrophy of the ventricle.
But it is probable that a large part of the dilatation
observed in chronic heart-disease resulting from acute
rheumatic attacks is not really due to the co-existing
mitral regurgitation, but that both dilatation and
regurgitation are persisting effects of the acute
attack ; the former due to its poisonous influence on
the cardiac muscle, the latter to the synchronous
endocarditis.   It has often been noticed in necropsies
on cases of " mitral regurgitation " that the apparent
damage to the mitral valve is far too slight to account
satisfactorily for the symptoms of cardiac failure.

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Probably in such cases it has played only a minor part in producing such failure, the main cause having been the persistent dilatation remaining after the original rheumatism. Thus it is easy to attribute too much importance to the presence of a systolic apex-murmur ; the prognosis depends far less on this than on evidence as to the size and strength of the heart.

The meaning and importance of a presystolic murmur in a child requires careful consideration. We may best arrive at the truth by carefully watching its development as the result of a rheumatic attack. The first indication of endocarditis is always the appearance of a systolic murmur at the apex, the second sound being still audible. In many cases it may be observed that after a time this second sound becomes doubled, so that the systolic murmur is followed by two second sounds of similar quality, which may be indicated by the symbol " whoo-ta-ta." This doubling is only heard in the apex region, it is not audible at the base ; it is, therefore, a different thing from the reduplicated second sound often heard over the base in cases of advanced mitral stenosis. No explanation of its production that I have seen has seemed to me quite satisfactory. I believe that the first of these two sounds is the normal second sound of the heart, compounded of the aortic and the pulmonary second sounds. This is caused, not by forcible closure of the aortic and pulmonary valves, but by the sudden extra strain thrown on the already

closed valves and on the aorta and pulmonary artery by the diastolic expansion of the ventricles. I think that the second of the two sounds, similar in quality to the first, is caused by the tension of inflamed and stiffened mitral flaps produced by the same rapid expansion of the ventricle. The observations of Ludwig and Hesse quoted by Dr MacAlister (*B.M.J.*, 1882), show that in the healthy heart during full dia-stole the mitral flaps do not " hang loosely down, but are stretched taut from basal ring to muscle-tip." The rapid expansion of the ventricle, caused by its elastic rebound after systole and by the rapid filling of the cardiac vessels, carries with it the papillary columns implanted in the ventricular wall, and thus through the chordæ tendineæ tends to fix and stretch the mitral flaps. If any sound is normally produced by this sudden tension of the mitral and tricuspid valves, it will coincide in time with the sound caused by the sudden strain on the aortic and pulmonary valves by the sudden diminution of pressure below them, and will thus form a part of the normal second sound. Now, if the mitral flaps are thickened by inflammation, they will be somewhat more slow to move, and it may well be that it will take a little longer to stretch them fully, and the sound produced by their tension will no longer accurately coincide with the sound of the strained valves at the root of the vessels, and may be so much delayed as to be distinguished by the ear as a second " second sound."

The first element of the double second sound at the apex never alters in character—it always remains a sharp short sound so long as it is audible at all. But the second element is liable to alteration; in place of a sharp short sound a short blowing murmur becomes audible. The descriptive symbol is no longer " whoo-ta-ta," but " whoo-ta-who." The time of this murmur in the cardiac rhythm would be described by some observers as " early diastolic," by others as " mid-diastolic." It is doubtless produced by a slight vibration of the stiffened mitral flaps caused by the commencing inrush of blood from the auricle, resulting from the suction-action of the rapid ventricular expansion. It will sometimes be noticed that the rhythm of the murmur seems to change when one listens over the cardiac septum, a little internal to the apex ; here the murmur may distinctly precede the systole instead of following it—it is presystolic. This must be due to the contraction of the auricle forcing a stronger blood-stream against the thickened mitral flap. At a later stage, there may be at the apex a systolic, immediately followed by a short early diastolic, murmur, or a short presystolic followed by a longer and louder systolic. It should be carefully noted that this presystolic is of a blowing character, and usually short. The combination of a short pre-systolic and a long systolic—both of blowing character, the systolic often the harsher of the two—is common in children after a rheumatic attack. It is usually

accompanied with evidence of great dilatation of the
heart.   It must not be taken to mean definite mitral
stenosis.   A necropsy shows a dilated heart, with or
without pericarditis ; and little or no contraction of
the mitral orifice, though the valve-flaps will be found
to be opaque and somewhat thickened and stiffened.
Occasionally the presystolic murmur becomes a little
vibratile, or very slightly rumbling ; it may end
sharply with the systole, or it may be prolonged
backwards and occupy the greater part of the diastole.
But it very rarely has the loud, rough, churning char-
acter of the presystolic murmur often found in
children over 15 or in young adults.   And the analy-
sis quoted above shows that marked mitral stenosis is
quite rare in children under 12 ; in the 150 necropsies
it was found only nine times.   The narrowing of the
orifice is a slow process ; it takes years for its full
development.

Aortic Murmurs.

Aortic murmurs are in young children much rarer
than mitral ones, and are of very little value in the
immediate prognosis.   They may indeed be accom-
panied by great cardiac dilatation, and by a sudden,
large, somewhat collapsing pulse.   But these pheno-
mena may be present in as great amount when the
aortic valves are unaffected.   The dilatation is of the
greatest importance as an element in the prognosis,
but any actual aortic regurgitation is usually very

slight and of no immediate importance. Care, how-
ever, should be taken not to put down to commencing
aortic disease a soft double sound at the base, which
may be the first indication of pericarditis. Such an
error is doubly unfortunate, for the exocardial lesion
is much the more serious of the two, and it also is
much more open to active treatment.

Conclusions.

To sum up, I would assert that the prognosis in
rheumatic heart-disease in young children is not to
be deduced from a consideration of the valvular
lesions believed to be present, except in the very rare
cases of advanced mitral stenosis ; but that it must be
founded mainly on three facts—the amount of cardiac
dilatation, the presence or absence of pericarditis, and
the evidence of a fresh rheumatic toxæmia, as shown
by sore throat, erythema, rheumatic nodules, arthritis,
and chorea.

AN ADDRESS ON ACUTE DILATATION OF THE HEART IN DIPHTHERIA, INFLUENZA, AND RHEUMATIC FEVER.

(Delivered before the Manchester Medical Society, October 1900.)

Sudden Death during and after Diphtheria.

IT is a fact of common knowledge that sudden death, often quite unexpected, is sometimes the result of an attack of diphtheria in a child. Most medical practitioners of ten years' standing have seen or heard of a case in which this has happened. Let me briefly refer to a few instances which I have myself met with, or which have been related to me by trustworthy observers.

CASE I.—When I was a clinical clerk, a young girl was admitted into hospital one afternoon on account of weakness after diphtheria. She sat up in bed, and did not seem ill. When I came next morning to take notes of her condition, she was dead.

CASE II.—Some years after this, when I was in charge of out-patients at St Mary's Hospital, I

admitted under my own care (by the kindness of Sir William Broadbent) a child suffering from diphtherial paralysis. I saw it in the ward the same afternoon, and perceived no cause for apprehension. When I came to visit it next morning, it was dead.

Case III.—A child of 3 years of age had suffered from diphtheria, but was thought to be convalescent. She was standing by a window, when some one entered the room a little abruptly. She turned quickly round, and fell dead on the floor.

Case IV.—A medical man in good practice attended a child for diphtheria. As she did not recover as quickly as he expected, he sent her to the seaside for change of air. She fell dead on the sands.

Cases V. and VI.—Another medical man had two little patients suffering from diphtheria. When one of them seemed convalescent, he allowed it to get up. The child was dead in an hour. Warned by this, he kept the second child in bed for a week longer; it was then allowed to rise. Within twenty-four hours this child also died.

Case VII.—A girl of nearly 11 years old had a severe attack of diphtheria. She looked pale after this, and was kept in bed for eight weeks. She was then allowed to rise. In five minutes after rising, she was dead.

Occurrences of this kind, so painful both to the surviving relatives and to the practitioner who has given a hopeful prognosis, call for careful study, to see if we can detect any indication which will warn

us of the coming danger and enable us to guard
against it.

Due to Degeneration of the Heart-Muscle.

On what do they depend ?   The first explanation
that suggests itself is that they are the result of a
neuritis of the vagus, causing arrest of the heart.   The
paralysis of the diaphragm or of the intercostal
muscles, which is sometimes produced by diphtheria,
may be due to a neuritis of the phrenic or of inter-
costal nerves, though it is not certain that this is
always the explanation.   But before we assume that
sudden death after diphtheria is caused by a neuritis
of the vagus, we must remember that whereas the
action of the respiratory muscles is brought about by
a stimulus from the respiratory centre in the medulla
oblongata, the action of the heart is maintained by
the automatic contractions of the cardiac muscle, and
that it is by no means certain that a neuritis of the
vagus would arrest the working of the heart.   We
must always bear in mind that the cardiac muscle
itself is the prime factor in the circulation, and it
seems likely that the fatal syncope which may follow
diphtheria is due to a diseased condition of the
muscular wall of the heart itself, rather than to disease
of the nerves which pass to it.   The microscope alone
cannot determine this question, for when degenera-
tion of muscular fibre is present it may be impossible
to say whether the toxic action was exerted on the

nerves, or on the motor end-plates, or on the muscle itself.

That the muscular wall of the heart does undergo serious degeneration in diphtheria, has been shown by several observers. Dr Sidney Martin * found that the diphtherial albumoses produced an advanced degree of fatty degeneration in the cardiac muscle, and he was unable to find any degeneration in the vagus nerve. Dr Mott † found fatty degeneration of the heart-muscle in each of four cases of diphtherial paralysis which he examined, but in only one of them was there degeneration of the peripheral nerves. My colleague, Dr Poynton, ‡ has recently published a comparative study of the condition of the heart-wall in diphtheria and in rheumatism respectively, and has described very marked degeneration of many of the cardiac muscular fibres, and complete destruction of some parts of them. It is therefore proved that in fatal cases of diphtheria the cardiac muscle is often much degenerated. The conclusion is irresistible, that it must be more or less degenerated in a large proportion of the cases which recover. Can we detect any evidence of this degeneration by clinical investigation? If so, fatal cardiac syncope might be avoided, and many lives might be saved which would otherwise be lost.

* Goulstonian Lectures, 1892.
† Croonian Lectures, 1900.
‡ *Lancet*, 12th May 1900.

The clinical indications which should be sought for are these :

1. Feebleness of the pulse-wave.
2. Feebleness and diffusion of the cardiac impulse.
3. Extension of the cardiac dullness to the left.
4. Feebleness of the first sound at the apex, with accentuation of the pulmonary second sound.

These four indications of a weakened left ventricle would all naturally be expected in a heart in which fatty degeneration of its muscle has been produced. They are all present, more or less, in many cases of diphtheria.

5. A fifth sign, which could not have been anticipated, but which is usually present also, is a marked accentuation of the aortic second sound. This is often very decided, yet the radial pulse is not tense, and one can only imagine that the tension in the aorta is raised by a contraction of the splanchnic arterioles through some central vaso-motor irritation caused by the toxins. If the vascular tension is much increased at the same time that the ventricle is weakened, the danger of fatal syncope is obviously great.

Physical Examination of the Heart.

All these physical signs need careful and accurate investigation. It is a matter much to be regretted that the examination of the heart is often very imperfectly performed, and that a hasty auscultation

—easily satisfied if no murmur is detected—is in too many cases the only method employed. But alterations in the normal sounds of the heart may be vastly more important than a murmur ; an almost inaudible first sound, or a greatly exaggerated aortic second sound, may be much more alarming. One must go even further, and say that the stethoscope has been far too dominant in the physical examination of the heart (and also of the lungs), and that palpation and percussion are too much neglected and often most inefficiently performed. Especially with regard to percussion, the ordinary method of examination of the heart is quite fallacious and almost useless. What is called the " superficial cardiac dullness " (often the only point investigated by percussion) informs us merely how much of the heart is not covered by lung ; it gives us no information as to its actual size. Yet, to be able to detect this actual size and to discover any enlargement of the left ventricle or of the right auricle, is often of the most extreme value to the practitioner, for a correct determination of these is in very many cases a matter of vital importance to the patient.

The difficulty felt by many in ascertaining the true size of the heart is largely caused by the word " deep." The outer margins of the heart are covered by lung, hence it has been thought that the true cardiac dullness can only be detected by a forcible percussion which shall bring out the deeper dullness in contrast

with the overlying pulmonary resonance. Unfortun-
ately this forcible percussion brings out far too much
pulmonary and gastric resonance from a distance, and
usually confuses the result. The trained ear may
sometimes be able to detect the change of note at the
same point, whether the percussion employed be light
or forcible, but a light percussion demonstrates the
margin of the heart with greatest certainty. The
edge of the heart on either side is thick and airless,
while the overlapping lung is thin, so that light
percussion easily reveals the limit of the heart to left
and to right, as well as the smaller area of its surface
which is not covered by lung.

A finger of the left hand is by far the best plexi-
meter, and it is one which is always available. The
terminal phalanx should be firmly pressed on the
spot where percussion is to be practised, and the rest
of the finger kept away from the chest-wall ; or the
middle phalanx may be used if the terminal phalanx
is slightly hyperextended, so that both it and the
first do not press on the chest. If this method be
adopted, the practitioner will have no difficulty, after
a little practice, in defining with very considerable
accuracy the limit of the heart both to left and to
right. The limits thus discoverable are verified by
post-mortem examination in fatal cases.

When the extent of the cardiac dullness trans-
versely has been determined by careful light per-
cussion in the fourth right, and the fourth, fifth, and

sixth left intercostal spaces, the position of the lateral margins of the heart may easily be defined if it be remembered that they both slope upwards and inwards, and the percussed finger be held in a position parallel to this slope in each case. The right margin above the nipple-level rapidly approaches the sternum ; but when the right auricle is much dilated, its dullness may be detected in the third space as well as in the fourth. The left margin normally rises to the inner side of the nipple, but in a moderately dilated heart the limit of dullness will be found to pass through the nipple, and where the dilatation is great it may cross the vertical nipple-line at one, two, or in extreme cases even three fingerbreadths above the nipple. For further details, I may refer to my paper on Acute Dilatation of the Heart in Rheumatic Fever in the *Medico-Chirurgical Transactions* for 1898.

Cardiac Dilatation in Diphtheria.

In a child suffering from diphtheria the cardiac dullness is usually increased towards the left. It is very important to determine carefully the extent of this increase. So long as it does not exceed one fingerbreadth outside the left nipple-line, there is, I think, usually no immediate danger. But if the dullness is greater than this, the case should be very carefully watched. If the dullness extends two fingerbreadths to the left of the nipple-line, there is

urgent peril, and the child must not be allowed to sit up in bed for any reason whatsoever.

I wish to draw special attention to the fact that the increase of dullness is sometimes very rapid: a further extension from one fingerbreadth to two may occur within a few hours. This acute dilatation is frequently accompanied by vomiting, and this symptom is an important danger-signal. It has long been recognised that when vomiting occurs in diphtheria the prognosis is bad, but I do not think that it is generally known that the vomiting in this disease is often a sign of an acute cardiac dilatation.

The symptom may be due to other causes, but in every case in which it occurs, especially if along with it there is increased pallor of face and a feeble pulse, the size of the left ventricle should be most carefully investigated afresh, even though percussion had been practised only a few hours previously.

CASE VIII.—A little girl under my care at the Hospital for Sick Children, who had suffered from a severe attack of diphtheria six weeks before, and whose cardiac dullness extended one fingerbreadth to the left of the nipple-line, was seized with vomiting which was ascribed to some pharyngeal irritation. But her marked pallor attracted attention to the heart, and it was found that the dullness had within twenty-four hours increased to two fingerbreadths outside the nipple-line. By great care and watchfulness her life was saved. I have seen other similar cases.

When the shock of the acute dilatation has passed off, or if it have occurred more gradually, the patients may look and feel well ; yet their lives are in urgent danger. It is, I believe, cases of this kind which come to an unexpected and tragic end. It is the apparent recovery, while the heart remains enfeebled and dilated, which misleads the practitioner who does not carefully percuss and palpate.

The cardiac dilatation of diphtheria may occur at an early period of the illness, even after only a few days. But a rapid dilatation, or a rapid increase of an earlier dilatation, may take place even after several weeks. In the case already narrated, it occurred six weeks after the onset. It is therefore necessary to keep a very careful watch on the condition of a child's heart for at least two months after a severe attack of diphtheria.

I have seen one case in which a permanent dilatation of the heart, apparently the result of diphtheria, caused dyspnœa, dropsy, and such imminent danger to life, that removal of blood by leeches was necessary. But I doubt whether the diphtherial dilatation is often permanent ; the less seriously damaged hearts probably again become of normal size, while the worst cases die ; for the mortality of " diphtherial paralysis " is certainly much greater than would be inferred from the statements of the text-books. It must be added that the virulence of diphtheria is more intense in children than in adults. Herein

there is a marked contrast to the behaviour of the disease of which I next speak—influenza ; for this is often highly dangerous to adults, while it affects children much less frequently and usually less severely.

Cardiac Dilatation in Influenza.

In influenza, rapid dilatation of the heart frequently occurs, to a greater or less extent, within a day or two after the onset of the disease, and it sometimes causes fatal syncope. If the heart be carefully examined, it will often, though not in all cases, be found distinctly enlarged towards the left, the impulse diffused and weakened, the first sound feeble, and the pulse wave also feeble. Here again, as in diphtheria, if the increase of dullness does not exceed one finger-breadth to the left of the nipple-line, there is probably little danger. But if there is a second fingerbreadth of dullness, there is real danger. And in this disease also the extension of dullness may develop rapidly.

CASE IX.—In one of the first years of the influenza epidemic, a young man was admitted into St Mary's Hospital under my care, suffering from influenza, and obviously very ill, with some evidence of pleurisy, but none of pneumonia. The cardiac dullness extended as far as one fingerbreadth outside the nipple-line. Fearing possible syncope, I asked the house-physician to direct the night-sister to give a hypodermic injection of strychnine immediately if the patient were taken ill during the night. A few hours after I saw him, an attack of syncope occurred. The strychnine was

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administered and the house-physician sent for. On examining the patient, he found that the dullness of the left ventricle now extended two fingerbreadths outside the nipple-line. This I myself found to be the case the next day. Forty-eight hours after the first attack of syncope, a second occurred ; the house physician found that the dullness now extended to three fingerbreadths to the left of the nipple-line. Half an hour later he was again summoned, and found the patient dead.

Whether acute dilatation may occur some weeks after the onset, as in diphtheria, I do not know ; but it is not unlikely, for many of the sequelæ of influenza do not manifest themselves until weeks or even months after the attack, and it is quite possible that an extra cardiac failure may be one of these. It is certain that the dilatation caused by influenza may remain as a permanent dilatation, and may give rise to very serious symptoms. Thrombosis is also of frequent occurrence after influenza, probably due in part at least to the enfeeblement of the heart. This thrombosis may in its turn produce pulmonary embolisms, resulting in great dyspnœa and hæmoptysis, or even in sudden death from obstruction of the pulmonary artery.

Minor degrees of cardiac dilatation after influenza may cause merely a feeling of incapacity for exertion. Unless the size and strength of the left ventricle be carefully ascertained, such a patient may be considered a hypochondriac, and very injudicious advice may be

given to him. Active exertion, unless carefully controlled, may do such a patient much harm by increasing the dilatation. Any prolonged strain—for instance, the effort of cycling uphill—may be very injurious after an attack of influenza.

CASE X.—Some years ago, I was asked to see a young lady whose only complaint was that she felt languid and incapable of exertion. She had suffered from influenza some months before. She was very fond of cycling, and had acted as the " hare " in games of " hare and hounds " by cyclists. Her left ventricle extended to two fingerbreadths outside the nipple-line. There was no murmur. By careful regulation of her exercise she recovered completely, and the limit of the cardiac dullness is now well inside the nipple-line.

Cardiac Dilatation in Rheumatic Fever.

In rheumatic fever, even in the most subacute attacks, acute dilatation of the heart seems to be invariably present. Since I first observed its occurrence, in 1894, I have never seen a first attack of this disease, whether in a child or in an adult, in which it was absent. When the rheumatic attack is over, the dilatation lessens and the cardiac dullness may again become of normal extent. It may be well to quote a recent case in illustration of this :

CASE XI.—A woman of 28, who had never previously suffered from rheumatism, was admitted into St Mary's Hospital under my care on 29th May 1900,

on account of some pain in her limbs, of five days' duration, with slight swelling of both knees and of one ankle, and some œdema of the legs. The temperature was 101° F. There was no murmur, but the cardiac dullness extended two and a half fingerbreadths outside the left nipple-line. The house-physician prescribed potassium citrate with caffeine and strychnine, but no salicylates. After twenty-four hours the rheumatic symptoms were worse, and the dullness had increased to three fingerbreadths to the left of the nipple-line. The medicine was then changed to sodium salicylate and bicarbonate. Within thirty-six hours the temperature had fallen to normal, and it remained normal. On 5th June—five days after the change of medicine—the left limit of the cardiac dullness was only one fingerbreadth and a half outside the left nipple-line. On 12th June it extended only one half fingerbreadth to the left of the nipple, and on 15th June the border of the dullness was in the nipple-line itself. On her discharge from the hospital the cardiac dullness and sounds were normal.

In this instance the rheumatic dilatation was unusually great for a first attack, and the cardiac weakness had produced some œdema of the legs, which is also unusual in a first and apparently slight attack of rheumatism. The latter symptom disappeared after three days' rest in bed, and coincidently with a marked improvement in the rheumatic symptoms and with the fall of temperature. Both the œdema and the considerable increase of cardiac dullness indicated a severe implication of the cardiac muscle, yet there was no evidence of endocarditis or pericarditis.

In later attacks also an acute dilatation is usually present, for it may almost always be observed, on careful examination, that the dullness of the left ventricle diminishes to some extent—greater or less —as the attack subsides. At all events, one may say with certainty that an acute dilatation of the heart is much more common in rheumatism, even in slight attacks, than in either diphtheria or influenza. Yet, though more common, it is far less dangerous. I have already said that an extension of the cardiac dullness to two fingerbreadths outside the left nipple-line is an indication of grave danger in a child affected with diphtheria. I will now add that the same amount of increased dullness in a child suffering from rheumatism implies, in itself, no immediate danger of death whatever.

It is a very remarkable fact that the dilatation of rheumatism is so much less dangerous than that of diphtheria or of influenza, in spite of its greater frequency in considerable amount. The difference must be produced by a different effect of the several toxins upon the cardiac muscle. In diphtheria, and apparently in influenza, the muscular fibres of the left ventricle suffer greater destruction; in rheumatism, the myocardial changes are less intense, and one can only suppose that the elasticity of the ventricle is more affected. Dr Poynton's sections show that, though in the rheumatic heart there is evidence of fatty degeneration of the cardiac muscular fibres, with

interstitial foci of small cells and vascular dilatation, yet the destruction of muscle is much less pronounced than in the diphtherial heart.

But though, as I have said, an increase of cardiac dullness to two fingerbreadths outside the nipple-line in a case of rheumatism involves no danger of sudden death, yet a further extension, occurring rapidly, may cause decided symptoms of collapse. Here again, as in diphtheria, a sudden vomiting is often the danger signal. Pallor, coldness, and general feebleness follow, and the cardiac dullness is found to have increased by an additional fingerbreadth within a few days or even within a few hours. From two finger-breadths outside the nipple it increases to three, or from three it may extend to four; at the same time the border of the left ventricle reaches a greater height above the nipple, from one to two finger-breadths, or from two to three. The cardiac impulse becomes more diffused and much weaker. Mitral valvulitis will almost certainly be present, causing a systolic murmur at the apex, and very frequently a presystolic murmur is present also. There may be in addition evidence of pericarditis or of pericardial adhesion. The implication of the mitral valve, allow-ing regurgitation into and therefore increased tension within the auricle, has doubtless some share in the great enlargement of the left ventricle which may occur in the later stages of rheumatism in a child. But the damaged condition of the cardiac muscle is

certainly the chief cause, and such cases should not be looked upon as simply " mitral regurgitation." In children the dilatation is of much greater importance than the valvular lesions. But it may easily escape the notice of a practitioner who does not constantly practise a careful examination of the heart by palpation and percussion, for it is very remarkable how slight may be the superficial evidence of a rheumatic toxæmia, which yet is able to produce a perceptible enlargement of the heart.

CASE XII.—A little girl under my care, who was convalescent from a rheumatic attack which had dilated her left ventricle to two fingerbreadths outside the nipple-line, was found one day to have some bronchial catarrh, with slight pain in her left ankle, and a new small nodule over this joint. On my next visit to the ward, I was struck with her pallor and evident illness ; the ankle was better, but the left ventricle was now dilated to three fingerbreadths instead of two.

CASE XIII.—In another child, the only obvious indication of a rheumatism which perceptibly enlarged his left ventricle was a very slight and transitory patch of erythema on his neck.

The slightest suspicion of rheumatism in a child should therefore lead to careful and repeated examination of the heart. Even in adults, much oftener than is generally recognised, it is fresh rheumatism that kills, breaking down compensation. It is important to notice that at the necropsy of patients who have

died from chronic rheumatic heart-disease, there is usually evidence of fresh endocarditis on the cardiac valves. And, clinically, it may often be observed that when a case of mitral stenosis breaks down there is some evidence of fresh rheumatism.

CASE XIV.—A woman recently under my care at St Mary's Hospital exemplified this. She was moribund on admission, and was found to have mitral stenosis, a much dilated heart, and thrombosis of the right jugular vein. It was thought by all who saw her that she would not live more than a day or two. Venesection three times repeated, once from each arm and once from the left jugular, saved her from the immediate danger ; the thrombosis did not extend, and she improved so much that she begged to be allowed to go home to look after her children. But during the five weeks that she lived after her admission there was persistent recurrence of a slight rheumatic arthritis in the joints of her hands—an arthritis which at once yielded to salicylates, but regularly returned when the medicine was omitted. At the necropsy evidence of fresh endocarditis was found, in addition to the mitral stenosis.

I have now shown that diphtheria, influenza, and rheumatic fever are all, in various measures, associated with acute dilatation of the heart. In fatal cases of diphtheria and of rheumatism, myocarditis and fatty degeneration, or even destruction, of cardiac muscle are often found ; probably similar lesions exist in influenza.

Now, it cannot be questioned that in diphtheria and

in influenza the injury to the heart is caused by the
deleterious action of the toxin produced by a microbe.
In each of these two diseases the microbe is well
known.   Is it not, then, in the highest degree probable
that rheumatic fever also is a microbic disease, and
that the resultiug cardiac inflammation is due to its
toxin?   A study of rheumatism in childhood compels
me to say that it is as certainly microbic in nature as
measles or whooping-cough, in neither of which the
microbe has yet been demonstrated.

But it is no longer a matter of inference merely
that rheumatic fever is a microbic disease, for it has
been proved by Dr Poynton and Dr Paine of St
Mary's Hospital* that a diplococcus can be isolated
from the tissues of a patient who has died from rheu-
matic fever, and also from blood obtained by vene-
section during life, which can be cultivated in pure
culture in a suitable medium, and which, when injected
intravenously into rabbits, can produce in them a
transitory arthritis of several joints, tenosynovitis,
pericarditis, valvulitis, cardiac degeneration and dilata-
tion, pleurisy and pneumonia, and yet nowhere any
suppuration.   By careful staining of their sections,
they have succeeded in demonstrating these organisms
in the substance of the mitral valve, in the tonsils,
and in many other organs.   They have found them
also in that most typically rheumatic lesion, the sub-
cutaneous nodule.   Their results will, of course,

* *Lancet*, 22nd and 29th September 1900.

require confirmation by other observers, but it certainly seems that the problem of acute rheumatism has at last been solved.

Importance of the Diagnosis of Cardiac Dilatation.

Finally, let me appeal for greater care and accuracy in the examination of the heart by percussion and palpation. It is a matter of great importance to the patient. Dilatation and feebleness of the left ventricle may indicate, as I have shown, urgent danger of death from syncope, and neglect of this indication may cost the patient's life. Dilatation of the right auricle (quite easily detected by percussion in the fourth right interspace), and weakness of the right ventricle (detected by palpation of the epigastric region) are usually accompanied by considerable dyspnœa and often by some lividity. A marked degree of dilatation of the right auricle (from two to three fingerbreadths to the right of the sternal margin in the fourth space) may indicate grave danger of death from asphyxia and call urgently for venesection or leeches.

After a considerable experience as an Examiner in Medicine, I am forced to the conclusion that these facts are still very inadequately recognised, for I rarely meet with a candidate who understands the object of percussion of the heart or the proper method of procedure. He usually thinks only of the useless "superficial cardiac dullness," and contents himself with trying to determine a horizontal upper limit

(which does not exist), and then palpating the cardiac impulse, as if that were equivalent to the left border of the dullness! The right limit of the heart he usually neglects altogether. And even some physicians of great eminence are apparently not conversant with the fact that the dullness of the right auricle normally extends one fingerbreadth into the fourth right space, and that its border can be quite easily detected by careful light percussion.

Let me assure you that there is really very little difficulty in the determination of the actual size of the heart, and that a small amount of careful practice will demonstrate to you what an enormous advantage both to your patients and to yourselves will result if you will accustom yourselves to consider this as an essential part of the investigation of every case to which you are called.

A PRESIDENTIAL ADDRESS ON THE HEART OF THE CHILD.

THE laws of the Harveian Society of London require that at the conclusion of his year of office the President shall deliver an address. I ask your attention, therefore, for a few moments to a short study of a subject too much neglected—the heart of the child.

"The child's heart," said Dr Sturges, in the Lumleian Lectures for 1894,* "holds as many secrets as the man's, and is even more deceiving." If this be so, it needs the more careful study, and (even more than the heart of the adult) demands accurate investigation and patient observation. Yet who thinks it worth while to spend much time in examining the heart of a child? Does every medical man investigate the condition of this organ in each child-patient as carefully as he does in an adult? In how many instances does he even recognise that it is his duty to examine it?

* *The Lancet*, 1894.

Doubtless he feels the child's pulse, but does he bestow much care upon the investigation of the heart itself? Does he always remember that heart - disease is frequent in early life, that its beginnings may easily be overlooked unless he is careful, and that his neglect to ascertain the exact condition may be disastrous to the whole of the child's future? Think of the amount of work which the child's heart will have to undertake. Calculating at the rate of one beat per second, which is below the truth, it will have to contract no less than 31,536,000 times every year. If it survive for 50 years it will have performed the enormous number of 1576 millions of beats. And if we adopt the estimate of Dr Leonard Hill in the second volume of Schäfer's *Physiology*, that the human heart performs work to the amount of 1000 kilogrammetres every hour, we find that in a year its total work will be 8,760,000 kilogrammetres, and in 50 years 438,000,000 kilogrammetres. Translating this into English measures we find that during these 50 years the heart will have to lift 1,500,000 tons to a height of one foot. That is the work which lies before the child's heart if it survive through a life of 50 years. How can it possibly accomplish the task unless it is thoroughly sound and well? Remember, also, that the child's heart is an organ incompletely developed. It has to grow to its full size and to minister to a growing body. Any disease, therefore, which damages it not only hinders its future work but impedes its development

and the development of the whole organism. From this point of view, the integrity of the child's heart is even more important than that of the man's.

The child's heart can be examined by the same methods as are employed for that of an adult. In some respects it is even more accessible, for in the child there is no excess of adipose tissue, no great muscular development, no large pendulous breasts, and rarely any emphysema. If the practitioner is rough and awkward, he may make difficulties for himself by exciting the child's fears, but anyone who knows how to deal with children can usually examine the heart of even the most "spoiled" child with accuracy. Impatient words, an ungenial aspect, rough manipulation, or cold hands, may make a young child cry and resist with all its might; but pleasant speech, a kindly manner, gentle ways, and warm hands will usually calm the most nervous child and allow of a complete examination, provided that the undressing is done by the mother or the nurse, and that she stands by the bed while the examination is made.

The chest being thus exposed to view, we notice at once whether there is any thoracic deformity or any prominence of the precordial region, and the position of any visible cardiac impulse. Having noted this, the first idea of most practitioners is to pull out a terrible looking instrument called a stethoscope, which probably appears to the child a sort of pistol, and doubtless is going to hurt him not a little. This early

recourse to the stethoscope is a great mistake for more reasons than one. Keep it in your pocket to the last. A child is accustomed to being handled ; you may use your hands to examine him as much as you like if they are only warm and gentle. He will not object at all to percussion if it be done very lightly and with the fingers only, but he will not tolerate the forcible hammering which some medical men call percussion, and those illusory instruments known as pleximeters will certainly make him cry. Fortunately, what the child allows is exactly what the physician ought to use, the lightest possible percussion. It is this, and not the heavy thumping, which is to tell you the precise size of his heart. But first warm your hand and lay it gently over the precordial region, that you may feel the cardiac impulse and ascertain its strength, and whether it is localised or diffused. This gives you information as to the strength of the left ventricle. Then shift your hand gently to the epigastrium, and notice whether any impulse of the right ventricle is to be felt there ; if so, either there is some congenital malformation or there is some disease of the lungs or left heart. Slide the hand further to the hepatic region and see whether it meets the resistance of an enlarged liver, and try very gently to feel its edge. A single gentle tap on a finger below the costal margin will confirm or correct your observation.

Next proceed to percussion, but begin with a clear idea of the object of this measure, and the way in

which it is to be carried out. What is the object of percussion of the heart ? To ascertain the superficial cardiac dullness, say the text-books. But what is the value of this result when you have obtained it ? Almost nothing, from the cardiac point of view, though it may give valuable information about the left lung. What we really want to know and what can quite easily be ascertained is the exact size of the heart. We can, indeed, determine little or nothing by physical examination about the size of the left auricle, and we can form only an approximate opinion of the size of the right ventricle. But the size of the left ventricle and that of the right auricle can usually be ascertained with very considerable accuracy. Let your pleximeter be the terminal phalanx of a finger of your left hand. Let this phalanx be pressed somewhat firmly, yet very gently, on the spot to be percussed, and let no other part of your hand touch the chest-wall. Thus you will avoid the conduction of resonance from elsewhere which is a fruitful source of fallacy. First select a spot in the mid-axilla and percuss here with the lightest possible stroke ; then gradually shift your percussed finger towards the sternum and you will easily recognise the change of note when the border of the heart is reached. For though the left lung partly overlaps the heart it is only by a thin layer, and the margin of the heart is thick and airless and does not require what is called " deep " or heavy percussion to recognise it. Indeed,

such percussion often defeats itself, for it introduces pulmonary and gastric resonance from a distance, especially if the whole of the percussed finger be applied to the chest-wall.   Expect to find the cardiac margin a little to the left of the position of the impulse, for though it is usual to speak of the " apex-beat," it is not the extremity of the heart which strikes the chest-wall, but a spot on the right ventricle at some little distance from that extremity.   The left border of the cardiac dullness in health extends a little to the left of the position of the impulse ; when the heart is dilated, this difference may amount to a fingerbreadth or even more.   (If anyone objects to the term " fingerbreadth " as unscientific, he may say instead " 2 centimetres " ; but he does not thereby make the measure more accurate, he only introduces a false idea of mathematical precision.)   Next determine whether the dullness extends to the left of the nipple itself, and if so to what amount in the nipple-acromial line.   Two points on the border of the left ventricle having been thus determined, the line connecting them should correspond to this border, and this can easily be verified by holding the percussed finger parallel to this line.   The difference in tone to the left and to the right of this line will then be obvious at once.   Next determine the size of the right auricle.   Very few percussors pay the least attention to this structure, and many seem to be unaware of the fact that the dullness due to it may

always be detected, even in the normal heart, in the fourth right intercostal space. The third space ought to be resonant quite up to the sternum, and in the fifth space the hepatic dullness alters the note, but in the fourth space the dullness of the right auricle is present for about one fingerbreadth in an adult and rather less in the child. When the auricle is dilated, it may extend to two fingerbreadths in the fourth space, and from a half to one fingerbreadth in the third. When the dilatation is very great, it may amount to three fingerbreadths in the fourth space, one and a half in the third, and may even be detected in the second. The accurate determination of the size of the right auricle is a matter of the greatest importance and often indicates at once the necessity for leeches to relieve distension. Yet there is no part of physical examination which is so universally and systematically neglected, and the patient is too often allowed to suffer cardiac distress when he might be relieved in a few minutes. The suffering due to a distended bladder a medical man would promptly relieve, but that due to a distended right heart is allowed to remain, because his percussion is not accurate and bleeding is out of fashion. Recently, when I advised a practitioner in charge of a case of pneumonia to try the effect of a few leeches, he replied that he would "see if they could be obtained in the neighbourhood." Evidently the distended right hearts of this gentleman's patients got no relief.

You have now determined the most important facts about the heart, its size and strength, and your stethoscope is still in your pocket. But by this time you and the child are on good terms, and you may quietly produce it without alarming him. It should be flexible and binaural; the rigidity of the old wooden instrument makes its use difficult in the child, and you are apt to press too heavily with it. Avoid the cumbersome instruments with a spring; all that you want is a small and simple ivory cup as a chest-piece, with two rubber tubes from it to two ivory ear-pieces. If these fit the ears properly, there is no need for any spring. This more simple instrument is better for your purpose, for it makes no artificial noises of its own and it is less alarming to the child. If the latter shows any sign of fear, it may be well to let him have it in his hands for a minute or two and look upon it as a new kind of toy before he is made the subject of its scientific application.

In auscultating the heart of a child, be on the lookout for murmurs due to congenital malformation. Very peculiar some of them are, and sometimes very puzzling. The most frequent, I think, is a systolic murmur, often very loud, which is loudest at, or just below, the junction of the left fourth costal cartilage with the sternum; it is probably often due to an incomplete cardiac septum. The next most common, perhaps, is a systolic murmur over the pulmonary artery, loudest at the second left cartilage and con-

ducted towards the clavicle, often audible in one or both supra-scapular fossæ. This indicates a congenital obstruction of the pulmonary artery. It is often impossible to diagnose the exact condition of a mal-formed heart by physical examination, and in some cases there may be no murmur at all. A patent foramen ovale, unassociated with any other malforma-tion, is probably of little importance, and is a doubtful cause of murmur. Congenital murmurs are systolic in time ; a presystolic or diastolic congenital murmur is exceedingly rare. Congenital malformations of the heart mainly affect its right side, which has the predominance in activity during intra-uterine life. After birth this side has much less proneness to disease than the left, though the tricuspid valve does not always entirely escape in rheumatism. But the most important affection of the right heart clinically is secondary to acute or extensive chronic disease of the lungs or to disease of the left heart. In all such conditions a careful watch should be kept on the amount of distension of the right auricle. Think of the tremendous strain on the right heart which a pneumonia causes even within two or three days. The thin-walled auricle becomes greatly distended, and the stronger ventricle shares to a less extent in the distension. If the auricle did not act as a reservoir, the ventricle would soon be over-distended and its action brought to a standstill. To some extent, then, the dilatation of the auricle is conserva-

tive, but if it exceed a certain amount the border-line which separates safety from danger, is easily crossed. Distress and dyspnœa are experienced, and the right ventricle has greater and greater difficulty in expelling its blood.   If at the same time its muscular structure is poisoned by pneumococcal toxins, we can easily understand that, even in a child with healthy heart, liver, and kidneys, a pneumonia is a process danger-ous to life.   It is true that the child has a much better chance than the adult of weathering the storm, yet its violence may be much diminished and the patient often very obviously relieved by applying a few leeches over his liver.   Not only are the distress and dyspnœa diminished, but the patient is enabled to sleep.   When the right heart is over-distended, sleep is much disturbed and may be impossible. Relieve the over-distension by a few leeches, and the patient falls asleep without any hypnotics.   Within a short time after the bleeding you will find on careful percussion that the dullness in the fourth right space has diminished, often by as much as a fingerbreadth. The relief will almost certainly last for two days, perhaps longer.   But do not forget to percuss out the auricular dullness every day.   If the pneumonia continues to increase, it may be necessary to repeat the leeches in two or three days ; in a few cases of exceptional severity (and pneumonia in children under 2 years of age is not infrequently fatal) it may be advisable to use them even a third time.   And in

considering the question of bleeding in pneumonia, remember that pallor of face and smallness of pulse are not necessarily contra-indications, for after the right side has been relieved the pulse will be stronger and the colour of the cheeks improved. You will observe this if you watch carefully the effect of the application of leeches in such a condition.

What has been said about the importance of carefully ascertaining the amount of distension of the right auricle in pneumonia applies also to certain other pulmonary affections. In acute bronchitis, in chronic bronchitis with an acute exacerbation, in whooping-cough with its mixture of collapse, broncho-pneumonia, and emphysema, and in an asthmatic attack, it is important to notice the amount of distension of the right auricle. It is true that the determination may be difficult if the anterior base of the right lung is emphysematous or consolidated, and it may sometimes be impossible when there is fluid in the right pleural cavity, but in the great majority of cases of pulmonary disease it can be accomplished. In all cases of disease of the left heart, especially when it has been injured by rheumatism, it is extremely important to note carefully the amount of distension of the right auricle. It is not usually possible to detect in a primary rheumatic attack an acute dilatation of the right heart, such as seems to occur invariably in the left ventricle. Nor is it dilated in a first attack of chorea, while the left ventricle almost

always is. Even when the rheumatic process has damaged the left ventricle sufficiently to interfere with its suction-action, and thus to raise the tension in the pulmonary artery, the only physical sign which reveals this is the accentuation of the pulmonary second sound. For a considerable time the right ventricle will succeed in overcoming the increased tension without affording any clinical evidence of hypertrophy or dilatation ; but if the primary rheumatic attack has been very severe, or if a relapse occurs (and to this rheumatic children are exceedingly liable, especially when they are deprived of salicylates) and there is evidence of pericarditis or of great dilatation of the left ventricle, then expect to find a gradual increase in the size of the right auricle. Watch it carefully. If the dilatation of this structure is rapid, dyspnœa and cardiac distress will probably be present, but a more gradual increase will only be revealed by percussion. As time passes, the heart accommodates itself as well as it can to its difficulties; and if the rheumatism does not recur, a condition of comparative comfort may be attained in which the right auricle is distinctly dilated but not yet grievously hampered by distension. The dullness in the fourth right space may amount to one and a half or two fingerbreadths, and yet there is no call for relief by leeches. It is, however, a condition of unstable equilibrium. A fresh rheumatic attack or a little over-exertion may turn the scale. Increased

dyspnœa reveals the greater strain ; the auricle dilates further, and its dullness may amount to two and a half or even three fingerbreadths in the fourth right space, one and a half in the third, and half a finger-breadth in the second. Before this the liver has become considerably enlarged. If you find it down to the umbilicus, or even lower, and the amount of urine passed be decreasing, there is no time to be lost. The condition is a dangerous one, but relief may be given by prompt venesection or leeching. The auricular dullness will then be found to diminish ; the liver also will be smaller, and the digitalis, which was producing little or no effect before, will manifest its action. Hypodermic strychnine will assist in restoring compensation. It is surprising to see how much relief may be given in the later stages of rheumatic heart-disease by treatment of this kind, and how long life may be preserved even when the condition of the heart is such that the child is incapable of exertion. But the fatal susceptibility to renewed attacks of rheumatism, incapacitating the heart still more, at last defeats the physician.

Before leaving the right side of the heart, I must call your attention to a systolic murmur over the tricuspid region which is not very uncommon in healthy children. It is a low, soft, short murmur, best heard about half-way between the left edge of the sternum and the nipple-line, and usually becoming inaudible at a short distance to the left of this line.

It is sometimes accompanied by slight irregularity of the heart's action. Its site of maximum audibility is lower than that of the congenital murmur already described, and to the right of that of a mitral murmur. It does not indicate any organic disease.

Let us now study the left side of the heart. The left auricle is inaccessible, but enlargement of the left ventricle (in which enlargement the right ventricle probably shares to some extent) can be easily determined by careful percussion, and the strength of the ventricular muscle can be fairly estimated by observing the force and localisation of the impulse.

Dilatation of the left ventricle being very common in children who are out of health, it is important to start with a clear idea of the position of the left border of the cardiac dullness in a normal child. Dr F. J. Poynton determined this carefully in 35 healthy public-school boys between the ages of 12 and 14 years. In 21 out of the 35 boys the left limit was about one inch internal to the nipple, in seven it reached a vertical line through the inner margin of the areola, in five it extended as far as the vertical nipple-line, and in two it passed this line by three-quarters of an inch and by one inch respectively. One of these two boys, however, was living in the medical attendant's house because he was delicate, the other had recently suffered from influenza. Thus we may conclude that in healthy boys of from 12 to 14 years of age the left border of the cardiac dullness is

found at about one fingerbreadth internal to the nipple-line. In a few it appears to reach this line, and this seems the more common in the younger children, especially before 7 years of age. In 45 cases of children under 12 years old in the surgical wards of the Hospital for Sick Children, Dr Poynton found the left limit internal to the nipple in 19, in the nipple-line in 18, and external to the nipple in 8. But of course these were not perfectly healthy children, for they were all inmates of surgical wards. We may conclude that in normal children the left border of the cardiac dullness is usually distinctly internal to the nipple-line, that it may sometimes reach it, but that it rarely goes beyond it. This must be carefully remembered when we investigate the cardiac dullness in a child who is suffering from disease, for it is so common to find the left limit from a half to one fingerbreadth external to the nipple-line that we may be led to consider it as normal, and its discovery to be of no importance. But this inference would be false in fact as well as in logic ; its commonness only proves that its causes are many. The cardiac muscle of the child is perhaps specially susceptible to the deleterious influence of toxins and poisonous products circulating in the blood.

The most characteristic instance of this is diphtheria, which is much more fatal in children than in adults, sudden death being by no means very rare after a severe attack of diphtheria in a child. The

fatal issue is usually caused by extensive fatty degeneration and destruction of the cardiac muscular fibres, such as Dr Sidney Martin found to be caused in animals by injection of the diphtherial albumoses. In and after an attack of diphtheria in a child there is usually enlargement of the left ventricle, the impulse becomes diffused and weak, and the first sound is short and feeble. If the dilatation amounts to two fingerbreadths to the left of the nipple-line, the danger of death is great, and the case should be very closely watched. And if the limit of the dullness of the left ventricle is only one fingerbreadth outside the nipple-line, it must be remembered that a rapid increase in the dilatation may occur, even six or eight weeks after the diphtherial attack, and may cause sudden and entirely unexpected death at a time when the practitioner is looking for convalescence and has given a hopeful prognosis. Influenza, also, may produce a rapid dilatation of the left ventricle which may be dangerous to life, but in this disease the susceptibility of the child seems to be less than that of the adult.

The pneumococcal toxin appears to affect the left ventricle less than that of influenza, and though there is usually some enlargement of the cardiac dullness to the left in pneumonia, it is much less than the enlargement of the right auricle, and one is often doubtful whether the enlargement to the left may not be due to dilatation of the right ventricle pushing the

border of the heart further to the left. The rapid recovery which often follows the crisis in pneumonia suggests that the poisonous influence of the toxin of the pneumococcus on the left ventricle cannot be very great.

In typhoid fever, a gradual enlargement of the left ventricle is usually to be detected, along with diffusion and weakening of the impulse : the first sound also becomes weak and short. The increase in size often reaches one fingerbreadth outside the nipple-line, and may amount to two fingerbreadths. During convalescence, the ventricle gradually returns to its normal size and its strength increases. In tuberculosis, the left ventricle is frequently moderately dilated. How far this is due to a tuberculous toxin and how far merely to general debility and anæmia, it is impossible to say. Even in debility and in anæmia there may be poisonous products of perverted metabolism circulating in the blood, and in renal disease it is quite likely that some of the increase of the dullness of the left heart may be due to toxæmia as well as to the influence on the ventricle of the raised arterial tension.

In acute and subacute rheumatism an enlargement of the left ventricle with enfeeblement seems to be invariable. It is the first indication of the effect of rheumatism on the heart, and may be detected when there is no evidence of endocarditis or pericarditis. The left border of the cardiac dullness almost always

extends beyond the nipple-line, even in the most subacute attack ; usually it reaches one fingerbreadth to the left of this line, and it may be even two finger-breadths to the left in a first attack of rheumatism in which there is neither rub nor murmur.   The impulse is diffused, and both it and the first sound are weakened.   As the attack subsides, the dullness tends to return to the normal limit.   It may do so com-pletely, but in many instances, especially when a murmur becomes audible, the left ventricle remains more or less dilated.   A second attack—and to this a rheumatic child is extremely liable—dilates it further, and an accompaniment of endocarditis is only too probable.   Pericarditis may occur in a first attack, but is usually a later phenomenon.   When it occurs it is always accompanied by great enlargement of the heart, causing an extensive increase in cardiac dull-ness, usually ascribed wholly to pericardial effusion. After repeated rheumatic attacks the child's heart may reach a size which is almost incredible to anyone who has not observed it in the wards and post-mortem room.   Clinically, it may extend four fingerbreadths to the left of the nipple-line and three fingerbreadths in the fourth right intercostal space.   In the larger rheumatic hearts there is usually a systolic apex-murmur, and frequently a presystolic or mid-diastolic also.   Evidence of pericardial friction, more or less extensive, is common.   Occasionally a diastolic murmur at the base gives proof of aortic regurgita-

tion. It has long been known that in children the cardiac manifestations of rheumatism often are far more pronounced than any other indications of the disease. There may be little or no arthritis, no soreness of throat, no eruption or only a very small patch of erythema, no subcutaneous nodules, no chorea or only slight nervous twitching, yet the cardiac rheumatism may be nothing less than deadly. If all who have to do with children would constantly bear this in mind, much suffering would be avoided and many lives would be saved. The slightest suspicion of rheumatism should lead to a most careful examination of the child's heart by palpation and percussion ; the practitioner who in this matter relies solely upon auscultation betrays a carelessness which is almost criminal, and shows himself unfit to be entrusted with the vital interests of the child.

In chorea, an enlargement of the left ventricle, with or without murmur, is present in the great majority of cases. The left border of the dullness almost always extends from a half to one fingerbreadth outside the nipple-line. This seems to be an additional confirmation of the essentially rheumatic nature of nearly all cases of chorea—one extra link in the chain of proof.

The extreme tendency to cardiac disease in rheumatism and in chorea seems to be not merely the result of the poisonous influence of a toxæmia. This, no doubt, is a part of the deleterious action produced

by rheumatism, and it may perhaps sometimes be the sole cause in a slight attack which recovers completely, but evidence is accumulating to prove that there is usually very much more than this—that actual inflammation of the muscular substance and fibrous structures of the heart exists, and that this is caused by the local presence of a diplococcal micro-organism. Dr Poynton and Dr A. Paine have demonstrated such organisms in the cardiac valves (with intact epithelium) and in the muscular wall. The same investigators have proved that pericardial fluid from a rheumatic child when injected intravenously into rabbits can cause in these animals all the most characteristic effects of rheumatism in the child. They have also obtained from a rheumatic nodule in a child, carefully excised with aseptic precautions, a most abundant growth of the typical diplococci in pure culture. In rheumatism, then, we are dealing with an inflammation of the child's most important organ caused by the local presence of a pernicious micro-organism.

The question of treatment at once divides itself into two problems: Can we by the administration of drugs directly destroy these microbes, or arrest their growth and increase, without damage to the child? and have we any means of directly repressing the cardiac inflammation?

To the first question I reply that in sodium salicylate, in adequate doses, we have a drug which seems

to be definitely antagonistic to the rheumatic process. The theory that it merely relieves pain is only possible when adults are the subjects of the disease ; in the child it is at once seen to be absurd. The best proof of the genuine efficacy of salicylate in arresting rheumatism is found in the great tendency to relapse when the drug is being administered in doses which are too small, and especially when it is too soon given up. There is a widespread impression that salicylate of soda is " depressing to the heart." What is really depressing to the heart is the rheumatic microbe, its works and ways, and some of its pernicious effects have been attributed to the salicylate. Children bear salicylate well, and it rarely causes in them the unpleasant aural symptoms which are common in the adult. It seems to be almost as necessary to a rheumatic child as mercury is to a syphilitic infant, and some children with great tendency to rheumatic relapse ought to take a small quantity of the drug daily for a long time. Sodium bicarbonate is another drug which seems to be certainly useful in rheumatism. It may be given in double the dose of the salicylate and along with it. My impression is that the acute dilatation of the left ventricle subsides more rapidly when the bicarbonate has been freely given than when the salicylate has been administered alone.

The question whether we have any means of repressing the cardiac inflammation may also be answered with confidence. Leeches will diminish the

congestion of the cardiac vessels, even when applied over the liver, by lessening the pressure in the right auricle and thus aiding the escape of the blood in the coronary sinus and the intra-cardiac venules, and the local application of an icebag most certainly represses the cardiac inflammation. These two remedies are of the greatest possible service to the rheumatic heart. Ice would be depressing to the normal heart, but when preceded by leeches and used with care it is the reverse of depressing to the rheumatic heart. It relieves the depression caused by the rheumatism, and under its use I have often watched the dilated ventricle diminish and the feeble diffused impulse become changed into a steady, heaving, local thrust. Digitalis is of little service in the treatment of rheumatic cardiac inflammation in the child ; its opportunity is later, when the inflammation has subsided and the mechanical effects of the cardiac lesions manifest themselves. Then it will work wonders.

When we pass from inflammatory conditions of the heart to the ventricular dilatation and enfeeblement caused by toxæmia, we see that leeches and ice are inapplicable. Digitalis is sometimes of service, but the hypodermic injection of strychnine is of still greater utility. Iron is of value in anæmic debility, but in diphtheria I rely mainly upon subcutaneous injection of atropine when danger threatens. I first employed belladonna in diphtherial paralysis more than twenty years ago on physiological grounds. It

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has subsequently been continually employed at the Hospital for Sick Children, and I believe I have seen it save many lives.   In cases of less urgency the drug may be given by the mouth, but where the dullness of the left ventricle extends more than one fingerbreadth to the left of the nipple-line it should be given subcutaneously, with a frequency regulated by the imminence of the danger.

THE PATHOLOGY AND TREATMENT OF CHOREA.

OUR subject to-day is the pathology and treatment of that common disease of childhood which is so inadequately and incorrectly described by the name "chorea." Inadequately, for there is much more than abnormal muscular movement in this malady. Incorrectly, for whatever else a choreic patient may do, there is a total absence of any rhythmic movement which might fairly be described as "dancing." It is most unfortunate that the name should wholly misrepresent the disease. Perhaps, when the pathology is completely understood, a better name may be invented. Meantime let us define our subject by excluding all varieties of hysteria, all forms of habit-spasm, and all irregularities of movement resulting from gross cerebral lesions, such as athetosis and "post-hemiplegic chorea." And in this Section we neglect the rarer chorea of adults and of old age, and

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concern ourselves only with the so-called " chorea " of childhood.

The most obvious fact in chorea is disorderly muscular movement, spasmodic, clonic, irregular, involuntary, with imperfect control and co-ordination. If we observe carefully the distribution of these involuntary movements, we find that it is precisely the muscles over which we have most voluntary power that are most affected in chorea—the muscles of the face, those of the tongue, and those of the hands and arms. These are the muscles which specially express emotion—by grimace, speech, and the movements of the hands and arms which naturally accompany eloquent or excited speech, especially in the more emotional races. It is precisely these muscles which are most affected in chorea ; the face, tongue, and hands are much more implicated than the proximal parts of the upper limbs, the upper limbs more than the lower, and the lower limbs more than the muscles of the trunk.

Respiration, over which we have a real though limited control, is affected much more than the action of the heart, over which we have no voluntary power whatever. The heart's action may, indeed, be irregular in chorea, but it is much less so than the respiration, and the heart often works steadily enough while the breathing is quite irregular. Choreic movements are often more pronounced on one side of the body than on the other, and in slight cases they may be limited

to the limbs of one side.   Choreic movements cease
when the patient falls asleep, and recur when he
wakes.

These facts, taken together, indicate unmistakably
a disorder of the brain, and specially of the motor
centres.   In the Rolandic area of the cortex much
space is allotted to the movements which are specially
under voluntary direction and control—to the face,
hand, and arm—less to the lower limbs in proportion
to their size, and comparatively little to the muscles
of the back and of the abdomen.   The relative
affection of the different muscular movements in
chorea corresponds to the cortex arrangement.

It is clear that in chorea these centres are in an
abnormally excited state, for if you ask a choreic
child a question, its hands will often reply before its
mouth.   Place the child at rest, with its arms and
hands outstretched on the bed, and wait for a quiet
interval when the choreic movements have ceased, or
are at all events much diminished.   Then ask the
child a question, however simple, its name, or the
number and names of its brothers and sisters.   Usually
the choreic movements recur at once in the hands,
often before the reply is uttered ; or if they have not
previously ceased, they become very rapidly much
more violent.   It seems that the outgoing impulse to
the speech-centre finds the adjoining centres of the
motor area abnormally excitable, and overflows into
them.   Sometimes the choreic movements follow so

quickly after the question that it almost seems as if the ingoing auditory impulse had reached them directly.

Similarly, if the child squeezes an object firmly with one hand the other hand usually becomes more decidedly choreic. In a case of hemichorea, a strong squeeze with the unaffected hand brings out additional movements in the choreic hand, while a squeeze with the affected hand produces little or no effect on the other. This seems to indicate clearly that the hand-centre in the cortex on the one side is more excitable than that on the other.

The motor centres are not only irritable, they are also weak. This weakness may be obvious from the first, but it is apt to become greater as the irritability subsides. The grasp is feeble, and the weakness of the whole upper limb may be so great as to deserve the name of paresis. When the hands and arms are outstretched, the child standing or sitting, there is usually a slight flexion of the wrist, apparently due to loss of tone of the special extensors,* and the voluntary extensor effort of the common extensor of the fingers slightly over-extends the first phalanges. It may be impossible for the patient to walk, to stand, to rise from the recumbent position, almost impossible even for him to lift one leg over the other.

* Dr Beevor showed, in the Croonian Lectures of this year, that no obvious contraction of the extensors of the wrist can be detected until the extensor of the fingers has to overcome a resistance of at least 3 lb., but the choreic hand seems to indicate a loss of at least a normal tonus.

In a severe case the weakness of the tongue and lips is great, so that feeding is difficult, and speech becomes too great an effort. The laryngeal muscles may share in this weakness. But often there seems to be something more than mere weakness of the muscular mechanisms involved in speech, for, though these muscles are never completely paralysed in chorea, speech may be altogether lost for weeks or even for months. I have seen several cases in which speech was impossible for six or eight weeks, and one case in which it was absent for eight months. This child made at last a complete recovery, and she was subsequently in the same ward for another but slighter attack of chorea, in which her speech was not impaired. In such cases it is impossible to resist the belief that the motor speech-centre is itself affected. Thus we have an indication that the muscular movements and weakness are not the only evidence of a disorder of the brain in chorea. This inference is often confirmed by the very first attempt at conversation with a choreic child, for even the most kindly question will sometimes provoke an emotional storm. The child begins to cry without any obvious cause ; this may pass away quickly, or the crying may become violent. When it has ceased, he cannot tell you why he cried, and he will sometimes acknowledge that there was nothing to cry for. Such attacks may be accompanied by great excitement, and even by hallucinations of vision ; they then fairly deserve to be called maniacal,

In the absence of emotional. attacks there is often a listless, vacant look on the face of a choreic child which in health was alert and intelligent. This may be due in part to weakness and loss of tone of the facial muscles of expression, but it is probably also the result of a mental dullness caused by the disease ; for mental dullness and loss of memory are often obvious enough. The answers to the very simplest questions in mental arithmetic are often absurdly wrong ; and when the child is asked to repeat the alphabet, one or two letters are frequently omitted. Alterations, also, in temper and disposition are often complained of by the mothers.

Whether there is impairment of the sensory activities of the brain, general or special, it is difficult to say. Choreic children are not often definitely hyperæsthetic, and marked anæsthesia is not a symptom of chorea, though it may occur in older children who are also hysterical. Possibly there is sometimes a general lowering of sensibility.

Thus it is clear that in chorea much more than the Rolandic area of the cortex is affected, and it is probable that the whole brain suffers more or less. Is the cord affected also ? If so, its affection is over-shadowed by that of the brain, as in tuberculous meningitis. But one phenomenon is observed which may indicate an extra irritability of the spinal ganglion cells—an increase in the briskness of the knee-jerks and a tendency to a prolonged extension of the knee

when the patellar tendon is struck.   Possibly also the atonic condition of the wrist-extensors may be of spinal origin.

Are the peripheral nerves affected?   Choreic children sometimes complain of flying pains in the limbs, away from the joints, which may possibly be neuritic.   In some cases of chorea the knee-jerks cannot be obtained, though it is not certain that this is really a symptom of the disease, or how, if a symptom, it is produced.   And the occasional occurrence of slight optic neuritis, mentioned by Sir William Gowers, must also be remembered.   It may be added that choreic children often suffer from constipation ; the cause of this is uncertain.

In chorea, then, there seems to be a disorder of the whole cerebral cortex, probably of the whole brain, possibly of the nervous system in general, sufficient to produce very definite symptoms, and often lasting for a long time.   Yet the disorder is not a destructive one ; it usually ends in complete recovery.   The pathological changes are, therefore, if organic, of slight intensity, and it seems probable that the morbid state may be largely due to a toxæmia.   Yet the fact that the symptoms may be very definitely localised suggests that the cause cannot be simply a toxæmia ; there must be something focal as well. The changes found post-mortem in the brains of the few cases which end fatally have been mainly confined to the vascular system — hyperæmia, dilatation of

vessels, perivascular leucocytes, thromboses, and slight hæmorrhages. Alterations in the nerve-cells have also been found by several observers. A recent investigation of two fatal cases by modern histological methods by Dr Reichardt of Chemnitz * showed small hæmorrhages, irregularly scattered, with collections of leucocytes, chiefly mononuclear, and dilatation of vessels, with perivascular small-celled infiltration, in many parts of the brain, in varying amount. No changes were detected in the ganglion cells, but there were areas of fatty degeneration of nerve-fibres ; in the spinal cord the parts most affected were the root-fibres, and the anterior and lateral horns, and the posterior columns—the anterior and lateral tracts were free. Cultures from the fluid of the cerebral ventricles of the first case gave no result, but " staphylococcus aureus " was obtained from the heart-blood. In the second case there were " strepto- cocci " in the cardiac valves, and a few colonies of " staphylococcus albus " were obtained from the brain.

If we look for clinical evidence of a morbid blood- state in chorea, we find that it is frequently accom- panied by manifestations of the toxæmia which we call " rheumatism." Any of the ordinary rheumatic phenomena in a child may accompany chorea— tonsillitis, arthritis, erythema, nodules, cardiac dilata- tion, endocarditis, pericarditis, pleurisy. The most

* *Deutsches Archiv für klinische Medicin*, 1902, p. 504. For this reference I am indebted to my colleague, Dr Batten.

frequent of these is cardiac dilatation ; for in chorea, as in rheumatism, the left ventricle is almost always too large, the left border of the heart extending beyond the left nipple-line, often as much as one fingerbreadth, and the first sound being feeble.   The next commonest is a systolic apex-murmur : whether this is always due to valvulitis is uncertain, but in many cases it is so undoubtedly.   In almost all fatal cases of chorea recent vegetations are found on the mitral valve.   A fairly frequent evidence of rheumatism is the rheumatic nodule, single or multiple.   The neighbourhood of the various joints, also the tendons of the hands and feet, the occiput, and the vertebral spinous processes ought to be carefully examined for nodules in all cases of chorea.   The other manifesta-tions of rheumatism must also be borne in mind, and special care should be taken not to overlook a peri-cardial rub.

With regard to the relations of rheumatism and chorea, I may repeat what I wrote in my article on rheumatism in Allchin's *Manual of Medicine :* " The relationship between the two affections needs further elucidation, but the more carefully they are studied the more intimate it is found to be.   Choreic symptoms of slight intensity are common in children suffering from acute or subacute rheumatism ; and when there is no definite chorea there may sometimes be seen the tendency to emotional disturbance—the causeless and transient fits of crying which are so

often observed in chorea. The onset of a severe chorea frequently follows two or three weeks after the occurrence of symptoms which, though slight, were definitely rheumatic. During an attack of chorea undoubted symptoms of rheumatism may manifest themselves. Many cases of chorea which have apparently at the time no connection with rheumatism suffer from an attack of that disease a year or two later. After making allowance for possible coincidences, the conclusion is irresistible that there is some very close connection between the two diseases, and that in many cases chorea must be looked upon as a definitely rheumatic symptom."

I draw special attention to the fact that chorea is often the first of a series of rheumatic attacks. If this be ignored, and the relationship of the two diseases be based simply on the precedence of rheumatism, the connection of the two will inevitably be understated. It occurred to me that it would be worth while to investigate carefully the amount of subsequent rheumatism occurring within a few years after an attack of chorea in children who had had no rheumatism previously. Dr Batten was good enough to investigate this point in the out-patient department of the Hospital for Sick Children.*

He found in 115 cases of chorea that whereas the percentage of previous rheumatism was stated to be only 32.2 per cent., three years later so many of them

* *Lancet,* 1895, ii. p. 1195.

had subsequently suffered from rheumatism that the percentage rose to 43.5, and three years later still it had risen to 53.2, so that the lapse of six years had increased the percentage by 21 per cent. And this is below the truth, for after the lapse of six years as many as 38 of the 115 children could not be investigated. If the same proportion of these missing ones had become rheumatic, the percentage would be increased, not by 21, but by 28 per cent. This added to the cases of " previous " rheumatism would give a total percentage of 60 per cent.

And even these figures are probably too low. When we remember that pain and swelling of joints, which alone constitute the ordinary idea of "rheumatism," are slight and infrequent in the rheumatism of childhood, and that its sole and yet conclusive manifestation may be a subcutaneous nodule, or a small patch of erythema, or a dilated heart with a soft mitral murmur, one sees that statistics which are to represent fairly the relation between the two diseases must be made with the greatest care. All the points mentioned by Dr Cheadle in his Harveian Lectures must be borne in mind. For these reasons, many published statistics on the connection of chorea with rheumatism greatly understate the facts.

The clinical evidence is in reality very strong, and we turn with interest to the excellent work of Drs Poynton and Paine on the bacteriology of rheumatism to learn whether they can throw light on the patho-

logy of chorea. I need not remind you that these observers, by a series of investigations, have isolated from the tissues in cases of fatal rheumatism, and also from the blood of rheumatic patients during life, a diplococcus which they have been able to cultivate, and which when injected intravenously into rabbits has produced in them all the symptoms of a violent rheumatism in a child, and that a recent subcutaneous rheumatic nodule, excised with aseptic precautions two hours after death, at once immersed in a suitable medium and cultivated for forty-eight hours in an incubator, was found to contain an exuberant growth of this organism without the presence of any other. In my judgment these results are decisive.

Accepting, then, the fact as now demonstrated that rheumatism is a disease caused by a diplococcus, and remembering its close clinical connection with chorea, we ask with interest whether this diplococcus is also responsible for the production of chorea. The evidence on this point is as yet scanty, but it is extremely suggestive. One of the rabbits injected with a culture of this diplococcus obtained by incubating pericardial fluid from a fatal case of rheumatism, developed symptoms remarkably resembling chorea in a child— clonic, irregular, involuntary, spasmodic, muscular movements, especially of the forelimbs and of the face, with a condition of " nervousness " such that it started at any sudden noise. This rabbit was killed, and diplococci were found in the lymphatic sheath of

the vessels in the pia mater and in the endothelial cells of the blood-capillaries penetrating the cortex of the brain.

A second fact is this. Dr Poynton cut sections of the brain of a fatal case of chorea which had been preserved for three years, and found diplococci in the cortex itself, also in profusion in the mitral valve. These, however, were not cultivated, so that it is not proved that these were the rheumatic diplococci. Yet if we take these two facts together, and remember that Dana, Apert, Wassermann, and others have isolated a diplococcus from the brain in fatal cases of chorea, it becomes more than probable that before long the demonstration will be made complete. Fatal cases of chorea are happily uncommon, and it will require some time before the point can be proved.

But if it has been shown that these diplococci are almost always found in the heart in fatal cases of rheumatism, and if they have actually been found in the blood during life, it seems certain that they must often reach the cerebral circulation. If their presence in the pial vessels and the cortex is the cause of chorea, we must look upon this disease as a microbic invasion of the brain, as in tuberculous meningitis. Why the result is so different in these two diseases must depend upon a difference in the virulence of the microbe, and the different measure of resistance on the part of the phagocytes and other defensive

mechanisms. In this respect some recent observations by Drs Poynton and Paine are of interest. They found that in the arthritis of rheumatism the fluid effusion is usually sterile ; that the diplococci are present in the synovial membrane of the inflamed rheumatic joint, but that they are there seized upon by the phagocytic cells, and that the diplococci contained in these cells are so much devitalised that they can no longer be cultivated.

Why the rheumatic diplococci should cause chorea in children and not in adults ; why some rheumatic children should become choreic and others not ; and why girls should be choreic three times as frequently as boys, must depend upon differences of individual power of resistance. Even if the actual presence of the micro-organisms cannot be demonstrated in the cerebral blood-vessels and membranes in fatal cases of chorea, it is still possible that the " irritable weakness " of the cortical cells may be due to poisoning by their toxin, just as the toxin of the diphtheria bacillus selects certain parts of the central nervous system. If the toxin alone is the active agent, the analogy would be with diphtheria ; if the organisms themselves are present in the cerebral membranes, the analogy would be with tuberculosis. A similar doubt exists with regard to the dilatation of the left ventricle caused by rheumatism — is it always due to the presence of the diplococci in the cardiac muscle, or may the toxin alone be sometimes effective ? The

decision of this question must be left to future research.

Are we, then, to say that chorea is "cerebral rheumatism"? Yes, if we add, "in the great majority of cases." But we must not make the statement absolutely, for other microbes and other toxins may perhaps affect the cortical cells in the same way as the rheumatic diplococci and their toxin, just as the bacillus coli and the bacillus enteritidis of Gaertner may produce a continued fever resembling that caused by the typhoid bacillus ; and, to say nothing of "senile chorea," the disease known as Huntingdon's chorea is found to be caused by multiple organic sclerotic changes in the cortex. It is even possible that the sudden emotional disturbance caused by fright may in some way disturb the nutrition of the cortical cells in a susceptible brain in a way similar to the altered nutrition caused by the rheumatic toxin. Sir William Gowers reminds us that throughout the animal kingdom the emotion of alarm has a direct effect on the motor centres essential to the safety of the animal alarmed. Cases of chorea really caused by fright, in the absence of rheumatism, are rare, but they probably exist ; the difficulty is to prove the absence of rheumatism.

I desire then to maintain that every case of chorea, however mild, ought to be looked upon as presumably rheumatic. And when we remember the danger of untreated rheumatism in a child, and the heart-disease

which it so frequently causes, we see how great an injury one may do to a child by neglecting a slight chorea. Many a mitral stenosis in later life, with its distressful years and its premature death, might have been averted if the practitioner who attended the case of slight chorea in childhood had treated it vigorously as rheumatic.

If, then, in the majority of cases chorea means a brain infected with rheumatic diplococci, surely the treatment which cures rheumatism ought to cure chorea. Yet it is not the general experience that much has been effected in this way. May not this be because the doses given have been too small? If one wishes to cure a cerebral syphilis, large doses of iodide must be employed. Acting on this idea, I have lately given to cases of acute chorea large and frequent doses of sodium salicylate, to which invariably has been added twice the amount of sodium bicarbonate. This addition of alkali I think of great importance, from more than one point of view. In a considerable number of cases this treatment has produced a very rapid improvement. The plan is certainly deserving of further trial in acute chorea. Success seems to depend upon the amount given daily, a further improvement sometimes following each increase of the dose. That the improvement is not due merely to the rest in bed, nursing, and feeding, has been proved in some cases by allowing three or four days to elapse before administering medicine. Some

cases grow worse if left untreated, others improve slightly.

The dose of sodium salicylate for a child of 6 to 10 years should be at first 10 gr., with 20 gr. of sodium bicarbonate. After two or three days the quantities should be increased to 15 gr. and 30 gr. respectively. After two or three days more they may, if necessary be increased to 20 gr. and 40 gr. These doses should be given every two hours during the day, and every four hours during the night, ten doses in the twenty-four hours. Thus the total amount of salicylate given at first is 100 gr. daily, increased to 150 gr., and finally to 200 gr.

A careful watch must, of course, be kept for any symptoms of salicylate poisoning, and especially for a peculiar deep inspiration simulating the "air-hunger" of diabetes. If this occurs, the medicine must be immediately given up, for it is a sign of danger. It is, however, a rare phenomenon. There is some reason for thinking that it is really an acid poisoning. The similar air-hunger of commencing diabetic coma may sometimes be arrested by large and frequent doses of alkalies, and the only fatal case of salicylic air-hunger that has come under my own observation had taken the salicylate without any additional alkali. Air-hunger began to show itself in one case which I treated with aspirin instead of salicylate, without alkalies, and disappeared when the drug was omitted. It seems to me very important that each dose of

salicylate should be accompanied by twice as much bicarbonate.

The unpleasant symptoms sometimes caused by salicylates in adults, the deafness and noises in the ears, the headache, the mental symptoms and delirium, are exceedingly rare in childhood. Occasionally vomiting is troublesome, but it may usually be overcome by suspending the treatment for a few hours and then beginning again with a smaller dose, which should be gradually increased. Pulse-failure occurring during the employment of salicylates, both in children and in adults, is generally not due to the remedy, but is caused by an acute rheumatic dilatation of the left ventricle—a common, but usually overlooked, phenomenon in rheumatism. Careful observation of the position of the border of the left ventricle, as ascertained by accurate light percussion, should be made daily. As to any general " depression " from the salicylate, in children it is usually quite absent ; indeed, it has been remarkable how much brighter and more lively the patients have become during the treatment. It should be added that the large doses of bicarbonate do not impair appetite or disturb digestion in rheumatic children, and that, in spite of them, the urine often remains acid for a considerable time. Albuminuria is not caused by the treatment above advised.

Every choreic child should, I think, be kept for a time completely at rest in bed, however mild the

chorea may be. If there is much tendency to excite-
ment, restlessness, or emotional attacks, complete
isolation is very helpful. A cot with padded sides is
necessary in severe cases, and the most careful and
efficient nursing is essential. It may be needful to
give in such cases hypnotic drugs, as chloralamide,
chloral, or bromide, the first night or two, to secure
sleep, but in many cases these can be avoided if large
doses of salicylate and bicarbonate are used. The
diet at first should be of milk only.

In acute cases this treatment is often very successful.
The time at my disposal will not allow me to give
details of cases, but I will briefly mention one as an
illustration.

J. S., aged 7 years, was admitted into St Mary's
Hospital on 3rd July, suffering from general chorea of
moderate severity. She was kept in bed for six days
without any medicine. During this period her chorea
became distinctly worse. On 9th July, the movements
being more choreic than on her admission, 15-gr. doses
of sodium salicylate with 30 of bicarbonate every two
hours during the day and every four hours during the
night, were ordered ; the total daily dose being 150-gr.
of the salicylate and 300 of the bicarbonate. This
treatment was continued for six days. Some vomit-
ing was caused by the abrupt introduction of very
large amounts of salicylate, and three or four doses
were omitted in consequence. On 15th July, after six
days of this treatment, the inspiration seemed to be
abnormally deep, and the salicylate was stopped en-
tirely, while the bicarbonate was continued. But
already the choreic movements had very much dimin-

ished. On 18th July hardly any could be seen, and on 22nd July the child was perfectly quiet, except that there were very slight movements in the fingers.

It is usually advisable to begin the treatment with smaller doses, which often cause no vomiting at all, but in this case a large initial dose was purposely employed, as a test, and the result was most remarkable.

I could give other cases almost as striking as this, and now feel confident that this is the most successful treatment for acute chorea.

In chronic cases one cannot be surprised if the effect of this (or any other) treatment is less certain, for even though the microbes may be destroyed and their toxin neutralised, the cortical cells may possibly remain for some time in a condition of irritable weakness. It is not always possible at once to get rid of an effect by removing its cause. Where the cortical centres have been severely affected, time may be an essential factor in their recovery. Prolonged rest and the gradual introduction and increase of the stimulus of voluntary action may be necessary. Yet even in chronic cases I have seen remarkable benefit. For instance, in a case treated recently by 10 gr. of salicylate and 20 gr. of bicarbonate every three hours, improvement to a certain extent resulted, but not a complete cure. Large doses of liquor arsenicalis were given for a few days, but no good result followed. Then the salicylate and bicarbonate were given again in larger doses, 15 gr.

and 30 gr. respectively, and very soon the chorea
had ceased entirely.

I do not desire to discuss the use of other drugs
in chorea. Arsenic, in large doses for a short time,
sometimes produces distinct improvement, but if its
use is continued for more than a week, there is
danger of producing arsenical neuritis. Dr Eustace
Smith has recently advocated the employment of
large doses of ergot. Dr John Thomson, of Edin-
burgh, tells me that he has had good results with
antipyrin. I say nothing of aids to convalescence,
such as massage, the cold douche, and the adminis-
tration of iron and of other tonics. What I desire
to urge is, that every case should be looked upon as
presumably rheumatic, should at first be kept in bed,
and treated vigorously, as for rheumatism.

P.S. (*March* 1904).—Since the publication of this
paper, I have had further experience in the admin-
istration of large doses of sodium salicylate in the
treatment of chorea and of rheumatism. I find that
in adults the amount given may sometimes be
increased with advantage to 400 gr., or even 450 gr.,
daily, in ten doses (each dose accompanied by twice
as much sodium bicarbonate), and that some cases
of recurrent subacute rheumatism cannot be cured
by smaller doses. I find also that, even where the
first employment of the salicylate causes vomiting,
tinnitus, deafness, headache, or delirium, it is almost

always possible to accustom the patient to the drug by waiting for a few hours only, and then giving a smaller dose. This can usually be soon increased, and quite large doses safely given before long.

In choreic and rheumatic children, I find that after a short time 300 gr. of sodium salicylate, and 600 gr. of sodium bicarbonate (in ten doses) are generally well borne, that they rarely cause vomiting, and that no depression follows. There is a marked improvement in brightness of expression and manner, and also improved nutrition.

But it must be clearly understood that each of these large doses has been invariably associated with twice as much sodium bicarbonate—*e.g.*, 60 gr. of this drug with each of the 30-gr. doses of sodium salicylate. Without this addition, there is danger of causing an acid-poisoning, the evidence of which is an "air-hunger" like that observed in diabetes; and this may soon be followed by a fatal coma. I recently saw, in consultation, a child suffering from slight chorea, who had been treated with 15-gr. doses of aspirin, given three times daily. After seven doses (105 gr. in two days) she became comatose. Large doses of sodium bicarbonate (a drachm hourly) were at once administered. When I saw her, about twenty-four hours later, the coma had quite passed away, and she answered questions readily, but the typical air-hunger still remained. The inspirations were very deep, and the air filled both lungs; the pulmonary

regions were resonant everywhere, and free from
râles. She recovered perfectly. This case proves
that aspirin is capable of producing the most
dangerous symptoms of salicylic poisoning. But in
the cases treated with large doses of sodium sali-
cylate and bicarbonate, no such symptoms have
been observed, though in many of them the large
doses have been continued for two or three weeks.

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